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(British Medical Association)

Second Session: Dunedin, February 3 to 10, 1927

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SURGICAL TREATMENT OF GOITRE.

By CARRICK H. ROBERTSON, M.B., B.S. (London), F.R.C.S. (England),
Auckland.

THE subject of goître has been so thoroughly discussed at many medical congresses in the last few years that it would seem there is no more to be said. Yet in spite of this medical publicity there are still medical men who regard an enlargement of the thyreoid gland as of little moment. More especially does this apply to simple adenomata in the gland. It is true that they may exist for many years and give rise to no symptoms whatever, but it cannot be too strongly emphasized that a great number of these cases will in the end seriously affect the heart.

The classification of enlargements of the thyreoid gland which we adopt is that of Plummer:

1. Colloid or simple goître,
2. Adenoma without hyperthyreoidism,
3. Adenoma with hyperthyreoidism,
4. Exophthalmic goître,
5. Cretinism,
6. Myxœdema,
7. Malignancy,
8. Thyreoiditis,
9. Anomalies such as syphilis, dermoid cyst.

Only the first four divisions will be discussed in this paper, as they are the ones of chief surgical interest.

The first in the list, colloid or simple goître, includes endemic goître of which there may be more than one form.

Certain areas of New Zealand bear an unenviable reputation for goître; these areas have been well and graphically represented by Professor Hercus at a former conference. This form of thyreoid enlargement usually starts in childhood and is often called adolescent goître.

It starts as a true hypertrophy of the thyreoid, a condition brought about in this country at any rate by an absence of iodine in the soil and therefore in the foodstuffs and water taken in by the individual. Iodine is a necessary ingredient of the thyreoid secretion, so that its deficiency induces the gland to produce more secretion in its endeavour to remedy the fault; the factory has to be enlarged. The administration of iodine in the early stages will nearly always effect a cure, but if the enlargement is allowed to continue, the gland becomes more colloid in character and in a few years becomes the colloid nodular goître so frequently seen in the goîtrous areas of New Zealand.

After a still further lapse of years (Plummer gives the average time as eighteen years), the goître becomes a toxic one.

The second heading, the adenomatous goître, is somewhat different in type in that the adenomata have a definite capsule. These tumours are prone to softening and internal hæmorrhages and may eventually form cysts in which no traces of the original solid character remain.

These cases like those in the previous class are prone to become "toxic" and when they have become so, sections cut through the whole gland show areas of hyperplasia in the tissues surrounding the adenomata. It is quite possible that the operation of removing the adenomata cures the toxic condition by relieving the pressure on the gland lobules and lymphatic sinuses which carry away the thyreoid secretion.

The chief action of the toxic agent in adenomatous goître is, of course, on the myocardium, but before heart signs can be demonstrated clinically, there is found to be an increase in the metabolic rate. One of the commoner serious heart signs, namely, fibrillation, can soon be demonstrated, but there is still quite a long interval, maybe of a few years, in which the removal of the adenoma will immediately and dramatically cure the heart disease. If the adenoma is not removed, the myocardial degeneration goes steadily on, so that there must come a time when the heart becomes permanently damaged and removal of the adenoma could only prevent further damage, but not cure the condition. However, it would seem to me as a surgeon to be a very difficult matter for the physician to decide just when the fibrillation of the heart has come to such a pass that no good can result by removing the source of the poison. Such a case as the following will illustrate my meaning.

I was asked to see a lady who had a toxic goître, with a view to operation. For nine months she had been unable to lie down in bed owing to dyspnœa from cardiac decompensation; she had lost twenty-five kilograms (four stone) in weight, had considerable œdema of the legs, a pulse rate at the wrist of 40, whereas at the cardiac apex it was about 140. If it had not been that the patient earnestly desired operation, I would not have ventured. As it was, the operation was performed; it was deemed advisable to do it without removing the patient from her bed and four-fifths of the gland were removed under local anæsthesia. She was very ill for two or three days after the operation, but during this time she received excellent nursing, with the result that a complete recovery followed. Within a

year she was earning her own living, having regained her lost weight, her pulse being normal and regular in rhythm.

So many cases of cardiac decompensation are due to thyreoid intoxication that in this country at any rate every cardiac patient should be thoroughly examined for thyreoid enlargement and although I personally have not seen a thyreo-cardiac patient who has not a palpable enlargement of the thyreoid, Lahey, of Boston, insists that many thyreo-cardiacs are without thyreoid enlargement.

The physicians will pardon me for apparently trespassing on their domain, but in talking of the effects of thyreoid adenomata on the individual, the foolishness of trying to keep strictly to our own departments, in other words divorcing medicine from surgery, is more than usually apparent.

Thyreo-cardiac patients are nearly always women over forty; they are often much pigmented and appear years older than they actually are. Another feature is that they do not have the excited, jumpy condition of Graves's disease, but on the contrary may be apathetic. The most interesting thing about them is the remarkable way in which they respond to surgical treatment of the thyreoid. Besides general toxic signs, adenomata may give rise to local pressure symptoms.

The thyreoid gland is tightly enclosed by the cervical fascia so that an adenoma, growing in this enclosure may cause distress by pressure on the trachea; distortions of this tube are common. It is said that simple adenomata may cause paralysis of the cords by pressure on the recurrent laryngeal nerves and that before operation the surgeon should examine every larynx to make sure the cord was paralysed before his operation and not after. It is rare for a nerve to be paralysed by a simple adenoma, for though there are many cases of husky voices associated with adenomata, they are more generally due to the excessive mucus secretion caused by tracheal compression or distortion than to sluggish movements of the cords.

Adenomata and their cystic degenerations were the first thyreoid enlargements to be treated surgically, the first operations were punctures with evacuation of contents and sometimes the tumour was shelled out from the gland. Nowadays it is recognized that the whole thyreoid should be exposed or palpated at the operation in order that smaller adenomata in other parts of the gland should not be missed; if they are, they will surely grow and cause a so-called recurrence. Occasionally a "wandering" adenoma is found; as a rule it is under the sternum. If this is not specially looked for, it is easily missed, for it may have no connexion with the thyreoid.

The third type of enlargement, namely, exophthalmic goître, is surprisingly frequent in this country. In the public hospitals of New Zealand during 1925, there were treated 503 cases of goître, 195 of these or 38·7% were exophthalmic in type.

In my own series the percentage of Graves's disease to other forms was twenty-eight.

Though Auckland is not one of the endemic centres of New Zealand, it is possible many of the patients may have lived for a time in one of these areas. It is not unlikely that the early changes in the thyreoid caused by living in iodine-free areas has some bearing on the high incidence of exophthalmic goître in this country.

Clinically patients with exophthalmic goître differ from those with toxic adenomata in the possession of two distinctive symptoms: (i) a peculiar restless, fidgetty condition, easily recognizable by the surgeon before he is even aware that there is an enlarged thyreoid to be seen or felt and (ii) the exophthalmos. The tachycardia, tremor, flushings and feeling of heat are common to both the toxic enlargements.

There are some other minor symptoms frequent in Graves's disease and not in toxic adenoma, such as a weakness of the quadriceps extensor muscle, making the act of walking up stairs a difficult procedure and certain alterations in the finger nails.

Before touching on the treatment of exophthalmic goître, I shall refer briefly to the work of Williamson as reported in his Arris and Gale lecture in 1925. He shows that the thyreoid gland has two functions, one the production of colloid, the other the production of secretion; the two products can apparently be manufactured by the same gland unit, but not at the same time.

The secretion, as opposed to colloid, is formed from cells which when active fill the spaces in the gland units or epithelial columns. In Graves's disease these hyperplastic cells fill the whole picture in microscopical section; no colloid is seen. This continuous activity induces the formation of fibrous tissue—the "strain fibrosis" described by Adami—which eventually locks up large quantities of secretion in the gland units. It also locks up whatever colloid is formed; both are therefore rendered unavailable for the purpose of function and circulation. He believes that the exacerbations of Graves's disease are due to sudden influxes of secretion directly into the blood vessels because it is not now able to escape by the normal channels, namely, the lymph ducts issuing from the hilum of the gland which run down to and traverse the thymus. He suggests that normally the secretion is modified in some way before reaching the blood stream by the thymus.

Without going more fully at this time into Williamson's researches, I have said enough to show how he finally arrives at a division of Graves's disease into primary and secondary, the primary being the excessive secretion from a young hyperplastic gland and the secondary being those cases of Graves's disease in which the normal afferent channels of the gland have become blocked by fibrosis.

These views have been brought forward as they seem to throw light on some questions of treatment. They justify the claims of the physician that early cases are properly treated by medical means, for the hyperplasia of the gland coming on in young people is probably the result of some general bodily disturbance which, by its calls on the thyreoid for extra secretion has stimulated its growth. If the cause of this increased demand can be located and

treated, it is obviously better than treating the thyreoid locally; for example, septic foci such as may be found in the teeth or tonsils, might be treated with advantage, although our experience in Auckland is that such treatment is of little avail. Unfortunately, in spite of early medical treatment or ignorance on our part of just what early medical treatment should consist, some of these patients, young girls in particular, become so toxic that in order to save life or save a too protruding eye from becoming ulcerated, it is necessary to remove a large part of the gland, by this means lessening the output of thyreoid secretion.

In support of Williamson's theory we know that Graves's disease in young girls with a large soft gland is not so satisfactory from a surgical point of view as the condition of those patients who have a longer standing history and a hard small gland. In the former case after removal of much gland, the pulse rate may not steady down for some months and worse still, the small remaining fragments of thyreoid tissue are apt to grow into large lumps and necessitate further surgical removals, showing that the underlying cause of the excessive thyreoid secretion has not been altered by removal of the gland. These are the cases in which it is well to warn the relations that one operation may not be a cure.

We are on much stronger ground surgically when we come to treat the condition termed secondary Graves's disease, in which either the Graves's disease has developed in an already diseased thyreoid or the condition of primary Graves's disease in which the thyreoid has become fibrotic. In both cases the gland holds accumulations of secretion and colloid in its meshes which cannot get out by the normal channels; the disease has become a focal one and surgical removal of a greater part of the gland is attended with excellent results.

The success of surgery in Graves's disease is largely due to the excellent technique developed notably by Crile and others in America and Dunhill and his followers in Melbourne.

The removal of such a goître until a few years ago was attended by a mortality which made the physician more than loath to send his patients to a surgeon. Many of us here can remember the time when a pool of blood on the theatre floor round the head end of the operating table was regarded almost as a normal condition during a goître operation. We now know that in Graves's disease especially much loss of blood is a very serious thing and unless the surgeon can be sure that his technique is sufficiently good to prevent this, he should not operate on these patients. Perfect hæmostasis and gentle operating are essential to a low mortality rate. The surgeon should endeavour to be

> As gentle as the stroaking wind
> Runs o'er the gentler flowers.

Another feature making for safety in operating is the exhibition of iodine for a short period before the operation and for a few days afterwards. It has long been known that to operate during a so-called crisis of the disease is a fatal thing to do. By crisis we mean those periods of the disease when the nervous symptoms are at their height and the curve of the metabolic rate at its optimum. Before the use of iodine as a preoperative preparation we were never quite sure that on the morning of the operation a crisis might not be starting; the excitement of the preparation was often enough to send the metabolic curve up to a dangerous height. It often meant that instead of doing an extirpation of the usual five-sixths of the gland, a smaller operation, such as ligation of the superior thyreoid artery, was all that could be undertaken.

Since the adoption of iodine preparation for exophthalmic goître, it has not once been necessary to perform a two-stage operation in a consecutive series of forty-three patients. It is not too much to say that the improvements in technique in performing these operations, chiefly introduced in the American and Australian clinics, together with the use of Plummer's iodine solution have reduced the operation of partial thyreoidectomy to a safety comparable to appendicectomy.

During the two years ending January, 1927, there has been no mortality and the operations have numbered one hundred and fifty-three, including forty-two for Graves's disease.

Before leaving the subject of the administration of iodine to exophthalmic patients it would be well to point out that it is essentially a preparation for operation and not a curative treatment. It should only be given preparatory to surgical operation.

The best time to operate is usually about a week after the course of iodine has been begun. At this time the metabolic curve will have almost reached the normal level, but in two to three weeks it rises and no amount of iodine will induce it to fall again, so that if advantage is not taken of the iodine remission in the metabolic rate, a golden moment may be lost. It may not be possible to get such a remission a second time.

The clinical effect of iodine on patients with Graves's disease is that it almost at once relieves the patient of those two symptoms peculiar to this form of thyreoid intoxication, namely, the fidgetty movements and staring eye. So great is this effect that many practitioners are now prescribing iodine by way of medical treatment; for the reasons given above this is much to be deprecated.

The radiological treatment of goître is to be discussed in a separate paper, but it may not be out of place to refer to the surgery of the gland when this treatment has not given complete relief. In cases where it has been found necessary to operate subsequent to X ray treatment, the fibrosis increases the difficulty of the operation in two ways: firstly, the tissues surrounding the thyreoid in the neck and even the skin itself are sometimes found so fibrotic that neat dissection requires a new scalpel every few minutes, the edge becomes so quickly turned. The resulting scar in the neck is also less satisfactory, for example in one case the place in which the drainage tube had been inserted, took six weeks to fill up, so fibrotic were the walls of the sinus. The second difficulty has to do with the amount of gland to be removed. Sections cut

from radiated thyreoids have all shown thick bands of fibrous tissue enclosing active hyperplastic areas. These areas have no doubt carried on the intoxication which has induced the patient to seek surgical relief, but if the usual one-sixth of the gland is left behind after operation, this fragment may become or is already so densely fibrotic that there is not enough thyreoid left to carry on the normal functions of the body. In other words there is danger of the patient showing symptoms of hypothyreoidism.

RADIOTHERAPEUSIS OF EXOPHTHALMIC GOITRE.

By C. C. ANDERSON, M.B., Ch.B., (New Zealand), D.M.R.E. (Liverpool),
Dunedin.

IN discussing the question of the treatment of exophthalmic goître I consider that the time has come when radiologists can safely take up the position that radiotherapy is indicated in this condition mostly as the main line of treatment, though in some cases it is only a subsidiary one. It must be clearly understood, however, that ordinary medical procedures, such as rest, the use of bromides and if necessary digitalis, are required at first together with the extirpation of septic foci if the best results are to be obtained. We learnt long ago that rest in itself causes considerable improvement in the condition of these exophthalmics and we know that rest enhances the action of the X rays.

The great advantage of this method of treatment is that the abnormal cells are so influenced that they return to normal function; it may be that the abnormal cells are replaced by normally secreting cells.[1] [2] Further it can be employed no matter how bad the condition of the patient; in fact it is these desperately ill patients who often give the most startling results; though, of course, it is the patients with the slight, early changes who provide the best series of results.

It must be remembered, moreover, that a certain number of fatalities occurs no matter what line of treatment is followed. Death occurs when the patient is operated upon, irradiated, treated by ordinary medical measures or not treated at all; just as death after operation is not considered a contraindication to further operations, so the infrequent deaths following radiation should not be ascribed to radiation *per se*.

There is, however, a certain type of patient who does not always react favourably to radiation treatment, and that is the patient with a diffuse goître of the colloid type. I do not, of course, refer to an ordinary case of parenchymatous goître for which radiation is useless. These patients probably give better results if operation follows after one or two applications of the rays; that is before the fibrosis which the surgeon ascribes to radiation therapy, has had time to become at all definite. The effect of radiation is to slow down the secreting activity of the gland and to lessen the amount of secretion to be liberated into the blood stream during operative manipulation, thus improving the patient's chances.

I am quite willing to admit that there is a disadvantage in radiation therapy of this condition and that is the time factor. Radiation therapy takes time and that is important when the bread-winner is affected. There the operation risk with its chances of an early return to work must be set off against the prolonged treatment of radiation, but it is important to remember that once the early stages of the treatment are passed, the capabilities of the patient can be tested and a gradual return to work made, with the result that those distressing postoperative neurasthenic symptoms which occur in a small percentage of otherwise successful cases, are avoided. I consider that radiation therapy is of value in patients, admittedly few, who are the failures of the surgeon; despite one or more operations the condition has recurred. Even these receive some assistance from the radiologist.

My own impressions of the results of X ray treatment of this condition have been gained in three separate schools. First of all at Liverpool Thurston Holland gives small, frequently repeated doses of X rays, often daily at first until a favourable reaction, that is, slowing of the pulse and lessening of the palpitation is obtained and then at gradually increasing intervals. His results were exceedingly satisfactory. He had had one or two deaths from acute exacerbations which could scarcely be laid at the door of the X ray treatment, but he could also show one or two cases of mild myxœdema which occurred amongst Jews.

Following this I visited continental clinics where intensive radiation was used. Here the practice was to give one-sixth to one-third erythema skin dose, filtered through a half millimetre of copper or zinc, the patient being observed until no further improvement followed, when the next dose was given. Usually up to six doses would be given in the course of twelve months and this was generally sufficient. I tried this method in Manchester with satisfactory results. Of nine patients six were well three years later and two others were so much improved that they were able to carry on their ordinary household duties successfully. Only one, a patient with the colloid type of goître, did not react satisfactorily and underwent operation. The above patients reacted much more favourably than those who were treated by the ordinary method of a pastille dose (fifteen centimetre or six inch spark gap, two millimetre aluminium filter) repeated at intervals of from fourteen to twenty-eight days, which was usually employed for Professor Murray's patients. Having a whole-time appointment and being in direct charge of both sets of patients I naturally had excellent opportunities for making comparisons.

Here in Dunedin until quite recently we employed a modification of this last technique, namely, ten milliampères, with three millimetres aluminium filtration, an alternate spark gap of nineteen centimetres (six and a half inches) and a focal skin distance of thirty centimetres (twelve inches). Each side of the gland was treated for four or five minutes and the ports of entry were arranged so that the thymus also received radiation, the early applica-

tions being for four minutes, the later for five minutes. Of my private patients out of twelve with exophthalmic goître four are well and four so much improved that they are able to carry out their ordinary duties with a fair degree of success; one of these operated upon is now well. Two did not improve, largely owing to the fact that they did not attend regularly for treatment, though one operated on since is greatly improved; two patients died, one in Australia of an acute exacerbation while away on holiday, the other had two treatments and then ceased to attend. Of four patients with toxic goître three are well and one was improved, but abandoned treatment before the full benefit was obtained. Of those patients who are well, the treatments varied from six to eleven in number spread over a period of twelve months. There are in addition to these some five patients who have commenced treatment during the past few months and who are improving satisfactorily. I have quoted the cases of private patients particularly as the hospital patients appear merely to visit the department on their way from the medical to the surgical side. Their end results appear to be very successful and there can be no doubt in my mind but that this is in no small manner contributed to by the benefit they received from their X ray treatment.

Latterly we have had a long awaited deep therapy (intensive therapy would be a happier term) machine installed and we have treated all patients with this. So far it would appear that our end results will be much improved. Our technique comprises a current of two hundred kilovolts, twenty milliampères at fifty centimetres focal skin distance, one half millimetre copper with one millimetre aluminium filter and four minutes' exposure (roughly one-quarter erythema skin dose). With these patients the whole thyreoid and thymic regions are covered by the one application.

Radium in my hands has not given such satisfactory results as X ray treatment and is liable to give rise to unpleasant telangiectases, but, of course, it finds its own place in the general scheme of things. In spite of the unpleasant effect that it has on the skin of these patients, it is very useful for treating the ultranervous patient who is terrified at the sight of an X ray table, but who does not mind the little box being applied to her neck. To my mind it appears much more reasonable to irradiate the whole substance of the gland uniformly than one particular localized spot; that is what I consider the weakness of radium.

To sum up I consider that the advantages of X rays lie in the infinite variety and shade of penetration and quantity of radiation which can be used. This allows of almost perfect variation in order to accommodate the treatment to the individual needs of the patient. Few treatments are necessary. The advantage of radium is that there is no cumbersome machinery and the patient can be treated where she desires; while if radion be used, she can undergo treatment while following her usual occupation. The disadvantages of X rays are that the cumbersome apparatus is liable to terrify the ultranervous patient and that the patient must come to the doctor; with radium they are the unpleasant telangiectasis which results in spite of very heavy screening and the great number of treatments that are required. Finally close cooperation between physician, surgeon and radiologist will show which cases are most suitable for operation, for radiation therapy or for both combined.

References.

(1) James Ewing: "Radium Report of the Memorial Hospital," page 255.

(2) M. Y. Nemenow: "A Contribution to the Biological Action of Röntgen Rays," *British Journal of Radiology*, page 302.

DR. BAKER MCLAGLAN (School Medical Service, New Zealand) opened the discussion with a few remarks on the mode of attack by her department on the goître problem. In schools there were three methods of attacking the question: (i.) by ascertaining the incidence, (ii.) by the result of the exhibition of iodine (iii.) by the correlation of goître with septic foci and by the finding and treatment of septic foci. She emphasized the fact that Dr. Hercus's remarks might lead them to suspect that the disease affected children chiefly, but this was a purely statistical point. She quoted figures to prove that the incidence of goître in limestone country was lower than in surrounding areas, for example in Te Kuiti (limestone) there was a low incidence; in Waikato (pumice and gravel) there was a high incidence. She also referred to the hereditary factor as being one that was important, but likely to be overlooked. A very large percentage of babies of goîtrous mothers had goître, but a goîtrous baby from a mother free of goître was unknown. More work was needed to discover how long hereditary goître persisted. Dr. Baker McLaglan then showed graphs and charts illustrating work on the goître problem and the favourable results obtained by giving small doses of iodine.

DR. E. CHAMPION (Ballarat) asked for a more detailed description of Dr. Stawell's method of administering iodine.

DR. J. GUTHRIE (Christchurch) agreed with the opinion expressed concerning the importance of the public health aspect of the goître problem. He advocated an education of the people as to the value of diet as a source of iodine. He recommended further consideration of the sources from

TABLE I.

No.	District.	Amount Iodine in 10 million parts of soil.	Percentage Incidence of Goître.
1	Bluff	20	21
2	Invercargill and Southland	20	35
3	Waimea Plains	6	32
4	Clutha Valley	4	41
5	Taieri Valley and Milton	6	30
6	Dunedin	32	19
7	North Otago	13	17
8	South Canterbury	3	62
9	Christchurch	9	64
10	Banks Peninsula	9	31
11	North Canterbury	6	59
12	Marlborough	3	38
13	Nelson	17	29
14	West Coast	12	53
15	Wellington	40	25
16	Hutt Valley	16	41
17	Hawkes Bay	9	36
18	Gisborne	13	34
19	Horowhenua	137	12·5
20	Palmerston	9	33
21	Wanganui	8	45
22	Taranaki	166	47
23	Taumarunui	14	37
24	Te Kuiti and Pio-Pio	25	11
25	Waikato and Piako Valleys	55	30
26	Taupo and Rotorua	10	28
27	Bay of Plenty	18	32
28	Auckland	118	11
29	North Auckland	53	5

which iodine was derived in the food of the people. The incidence of goitre depended on the absence or presence of iodine in the food, food consumed being the source of body iodine. Sea food was especially rich in iodine, but in New Zealand this is scarce and of high price and inland people did not obtain fish readily. He suggested that the Government should help in the distribution of sea food especially in inland districts. Goitre was rare among Maoris who were great fish-eaters.

DR. A. C. B. BIGGS (Balclutha) inquired if McCarrison's work was not now accredited. He thought it should still be considered. He would like to ask Dr. Hercus if the conclusions from such good work were not to stand.

DR. T. L. PAGET (Stratford) stated that goitre was prevalent in Taranaki and that the province was not goitre-free. He had come to this conclusion after twenty-seven years' practice in that province.

DR. D. L. SINCLAIR (Hawera) disagreed with Dr. Paget and agreed with Dr. Hercus that Taranaki was comparatively goitre-free.

DR. MARY DE GARIS (Geelong) held that there was a combination of causes in the production of goitre, not only an iodine deprivation, but also a common toxæmia. She inquired whether it was known when goitre had become a national problem in New Zealand.

In the treatment of goitre Dr. De Garis had found that the exhibition of *syrupus acidi hydrochlorici* and "Iodex" ointment gave great benefit in early cases even if the focal sepsis in situations such as the teeth, antrum, tonsils and so forth were not removed. It was, however, more useful if sepsis were dealt with at the same time.

DR. D. GIFFORD CROLL (Brisbane) asked Dr. Hercus to elaborate the treatment and prophylaxis he recommended as preferable.

DR. L. E. BARNETT (President of the Congress, Dunedin) said that goitre was of intense importance in New Zealand. There was a possibility of doing something definite and helpful along public health lines. A small committee should be formed to draw up a remit to submit to general meeting in connexion with goitre prevention.

DR. HARVEY SUTTON in reply agreed that there were other factors in goitre causation besides the presence or absence of iodine in the soil. He agreed that more information was needed about the translation from simple to toxic goitre. The subject would be further dealt with in another meeting. He had experienced difficulty in collecting information on the point. He had not yet seen the simple goitre display toxic symptoms requiring sick leave.

He emphasized the fact that goitre prevention must be along physiological lines. The diet factor must certainly be considered. In the administration of iodine more work by age groups was needed and would produce interesting results. He thought that iodized salt was certainly the most fertile of possibilities in prophylaxis.

PROFESSOR HERCUS replying to Dr. Stawell, who had had uniformly unsuccessful results with the iodine treatment of simple goitre, said that this was not the New Zealand experience. He referred to Dr. Baker McLaglan's results. Salt iodized according to exact calculation of physiological requirements was a good preventive measure if used for all culinary and table purposes. In the cooking of green vegetables about two-thirds of the iodine were extracted by the water used, hence the water from vegetables should not be discarded, but used. The average daily intake of salt from all sources was about five grammes; this daily intake if of salt iodized to contain one part of potassium iodide in 250,000 parts would be an easy and biological way of supplying the iodine deficiency. This small amount would suffice for prophylaxis. It was wise to think of iodine in terms of milligrammes and not of grains. The daily iodine requirement of the healthy thyreoid gland was about forty milligrammes. If one grain was prescribed it was far too much iodine and the excess was promptly excreted in the urine. Any further increase in dose was similarly excreted.

Dr. Harvey Sutton had suggested that age group treatments would be a useful study. Already in Dunedin age group treatments had been carried out, salt iodized one part in 250,000 being used. By working among girls from two to three years up to 'Varsity women notable results had been obtained.

One speaker had referred to secondary causation. The dietetic was an important secondary factor, so also was focal sepsis. For example, after removal of septic foci, goitre had been known to decrease. But the primary factor in ætiology was iodine deficiency. McCarrison had recently modified his former views.

Dr. Hercus pointed out that Stratford, New Zealand, was on a gravelly plain with less humus and therefore less iodine and more goitre than the rest of Taranaki. In local observations the movement factor of the population was important and had to be kept in mind. In referring to sea fish as a source of iodine in food, he drew attention to the fact that edible seaweeds were preferable, while the white-fleshed fish, such as groper, so commonly used, was of low iodine content. The shellfish type were rich in iodine, for example oysters. Sea fish should be used more, but there were other elements in food rich in iodine, such as eggs, milk, leafy vegetables.

The universal iodizing of salt had been objected to on the grounds that it was an unjustifiable interference with the people's rights; but the method had been evolved from the careful biological study of the goitre problem and no dangers were incurred. If this method were adopted, the State should take strong action against the manifold goitre remedies. The Swiss Goitre Commission had long ago realized that in Switzerland. Professor Hercus hoped that a committee would be set up to go into the matter of the prophylaxis of goitre.

DR. BARNETT then proposed a committee to form a remit suggesting legislation in regard to goitre prevention. The meeting agreed to a committee of five: Dr. C. E. Hercus (convener), Dr. Harvey Sutton, Dr. S. S. Argyle, Dr. M. H. Watt, Dr. R. R. Stawell.

TABLE III.—CORRELATION WITH SEPSIS OF MOUTH AND THROAT.

Condition of Thyreoid Gland.	Ages 5-10 years.	Ages 11 to 14 years.
Without Goitre	30%	37·4%
Incipient Goitre	31%	37·1%
Small, Medium, Large Goitre	33%	51·4%

CORRELATION WITH POOR GENERAL CONDITION.
Children classed as "fair," "indifferent," "delicate" and so forth:
200 children without goitre included 24 (12%)
500 children with goitre included 17 (3·4%)

TABLE II.—SHOWING HOW SURPLUS IODINE IS STORED AND OFTEN CONTINUES TO ACT AFTER TREATMENT IS STOPPED.

Date of Examination.	Standard.	Enlargement of Thyreoid Lobes.			Measure in centimetres.	Remarks.
		Middle.	Right.	Left.		
May 8, 1922	P. 1	Small	Medium	Medium	26·2	Received between 5·22 and 4·23, a total of 30 grammes of sodium iodide.
April 10, 1923 ...	P. 3	Incipient	Small	Incipient	25·6	Improvement evidently the result of a year's treatment.
February, 1924 ..	P. IV	Incipient	None	None	25·8	This continued improvement I attribute to iodine stored as a result of a year's treatment.
May, 1925	S. II	Incipient	Bordering on Small	Incipient	26·3	Store of iodine exhausted and goitre begins again to enlarge.

Section VI.—Ophthalmology.

THE CAUSES OF CONCOMITANT STRABISMUS.

By A. M. Morgan, M.B., B.S. (Adelaide),
Adelaide.

I wish first to thank you for placing me in this honourable position and to ask your indulgence of my temerity in addressing this gathering of members of our branch of the profession, many of whom I am only too conscious are far better able to perform the task than myself, but you will perhaps be somewhat consoled by the assurance that my address will, at any rate, have the merit of brevity.

I have chosen my subject partly because I have never been satisfied even as a student with the text-book explanations of the causes of concomitant squint and perhaps for that reason it has always had a peculiar fascination for me, and partly because of the great frequency with which not only ophthalmic surgeons but general practitioners are consulted for this complaint, for it is one of the commonest with which we have to deal.

In 15,389 patients of all ages I found 925 cases of concomitant squint or 1 in 16·63 of all ophthalmic cases. In private practice the ratio was 1 in 23·19 and in children's hospital practice 1 in 6·71.

As a student I was taught Donder's theory of the causation of squint with which, of course, you are all familiar. Even at that time I was far from convinced of the completeness of that theory, for in the first place, were it correct, all hypermetropes should be afflicted with convergent and all myopes with divergent squint, which fortunately is far from being the case. Moreover, convergent squint with myopia and divergent squint with hypermetropia would be impossible, as would any kind of squint in an emmetrope or vertical squints which no kind of error of refraction can possibly influence.

Worth's theory[1] of the weakness or absence of the fusion faculty as a determining cause of squint explained some of these points. It explained why all people with errors of refraction do not squint, but not why hyperopes sometimes have divergent squint and myopes sometimes have convergent squint, nor why vertical squints occur. Nor does it explain why all squints are not cured by a full correction of the refractive error as theoretically they should be, nor why we may have a squint in any direction without any error of refraction at all.

Another fact which refutes this theory, is that the amount of deviation rarely corresponds with the amount of the refractive error. With an interpupillary distance of fifty-seven millimetres, which is the average among seventy-eight children between the ages of five and eleven years, one metre angle of convergence would equal 1° 35″ of deviation for each eye or 3° 10″ for both eyes; therefore a squinting patient with one diopter of hypermetropia in each eye should have a deviation of 3° 10″, no more and no less, whereas he very commonly has more. Supposing him to have 25° of squint, then 21° 50″ of that deviation must be due to some cause other than the error of refraction; this other cause in such a case is esophoria. If such a child have his error of refraction corrected, we cannot expect an alteration in his deviation of more than 3° 10″ and in practice this is found to be so; for, of course, the correction of his hypermetropia cannot have any effect upon his heterophoria.

An illustrative case is that of J.H.G., aged four years and seven months, who has an alternating convergent strabismus of 51°. His error of refraction is the same in each eye, namely $\frac{+2{\cdot}00}{+0{\cdot}5 \text{ at } 90°}$ His deviation should therefore be 7° 55″, leaving 43° 5″ unaccounted for. With glasses his deviation is 46° or a little more than it theoretically should be. Those patients who are found upon measurement to have approximately the amount of deviation that can be accounted for by hypermetropia, will generally have their eyes straightened by a full correction of their error; one can form a very accurate prognosis as to the likelihood of glasses being partially or entirely successful by calculating the amount of deviation which should be due to their hypermetropia. It is not necessary for a successful result that the amounts should exactly correspond; when such is not the case, the ultimate result will depend upon the keenness of the fusion sense which varies within wide limits even in adults and probably more so in children. All squinters, at any rate at the beginning, have some desire for binocular vision and in many there is still a remnant of this desire left even many years after the squint has become constant. This may perhaps explain why convergent squints of less than 10° are uncommon; the effort of correcting such cannot be very great and if the desire for binocular vision be greater than the effort required to render the eyes parallel, there will be no squint. If on the other hand the effort for correction be greater than the desire for fusion, then a squint will result. Therefore if we can nearly correct a squint either by glasses or operation, we may thereby bring the needed effort within reach of the desire and the remaining portion of the deviation should be cured. This also explains why an operation for squint, when successful, so often results in perfect alignment of the eyes, for there is no method by which the effect of a tenotomy can be regulated and I believe there are few operators who can regulate an advancement so accurately as to correct a squint of 5° or less; at any rate I am certain I cannot be sure of doing so myself. Nor do I believe that it is necessary to do more than to bring the effort for correction within reach of the desire for fusion and the eye will become straight. If this be so, it would not be necessary for the development of a squint that there should be any defect at all of the fusion sense. One can easily imagine a patient with so pronounced a heterophoria that even with the keenest of fusion senses the effort to correct it may be greater than the desire for binocular vision. A disproportion between desire and effort also explains why squint so often first manifests itself after an illness or injury, because the general debility following such an illness makes it difficult to overcome a heterophoria which before was easily corrected; the effort has become greater than the desire and a squint results. The suppression

and ultimate weakening of the desire for binocular vision may soon make such a squint constant. Whooping cough, because it is one of the most debilitating of the diseases of children, thus becomes a very common exciting cause of squint. Also it is quite usual to be told by the mother of a small patient that the eyes only turn when the child is tired or out of sorts, showing again that the effort has temporarily become greater than the desire; as the child's health and vigour improve, the squint disappears again for a time. Here again the repeated suppression of the desire for binocular vision frequently results in its ultimate loss and a constant squint developes.

What are the factors which cause the eyes to deviate from the parallel? And what part does the error of refraction play in the result? I think not so much as is generally supposed in convergent and none at all in divergent squint. To produce a convergent squint of 25° a hypermetropia of more than eight diopters in each eye would be needed, a condition which, to say the least, is more uncommon than squints of 25°; as a matter of fact it is not the highly hypermetropic patient who most commonly developes squint as should be the case if hypermetropia were a dominant cause. Moreover, there is a large number of squints in which the error of refraction could not possibly be causative, as divergent squints with hypermetropia, convergent squints with myopia and all squints in emmetropes and all vertical and rotatory deviations. Also the majority of convergent squints and all divergent squints fail to correct themselves under an anæsthetic, although there can then be no desire for either accommodation or need for convergence; the defect in such cases cannot be due to any cause other than heterophoria. An error of refraction may even be instrumental in partly or entirely correcting a squint. I have an emmetropic patient with a considerable divergent strabismus who can make his eyes parallel by exercising his accommodation, but as he can then not see clearly in the distance nor maintain the excessive accommodation for any length of time, the correction is of no use to him. Hypermetropia may, I think, in a limited number of eyes be the only cause of convergent strabismus; such eyes have usually a high error, the squints are often only occasional and are readily cured by the use of glasses alone. If then the error of refraction be only a minor factor in the causation of squint, we must look for the predominating cause in heterophoria. Of the 925 cases of squint mentioned above 738 were convergent and 187 divergent or a ratio of divergent to convergent of 1 to 3·94, which would seem to indicate that esophoria is nearly four times as common as exophoria, which taking all ages is far from being the case. In a series of 1,766 patients, of all ages from five to seventy-five, tested qualitatively for heterophoria (a few of these were tested quantitively as well by the occluding bandage, but not enough for a separate table) there were 633 with esophoria, 543 with exophoria and 592 with orthophoria or a proportion of exophoria to esophoria of 1 to 1·16. Marlow in his seven hundred patients tested quantitively found a great preponderance of exophoria;[2] there were 544 with exophoria to 93 with esophoria or a proportion of esophoria to exophoria of 1 to 5·84. His patients, however, were all adults except two and were all persons in whom there was reason to expect that there would be some considerable amount of latent deviation. If the patients in my series be divided into two age groups, five to ten (165) and eleven to seventy-five (1,601), we find a very different proportion, for in the first group we get exophoria to esophoria 1 to 2·45 and in the second group 1 to 1·06. Unfortunately it is not possible to get reliable results from patients under five years of age and only in the more intelligent of them; many subjects up to the age of seven had to be rejected as undependable. However, there is evidence that esophoria is still commoner under that age; for those occasional squints which are so often seen in infants, are almost invariably convergent. If an eye be blinded in infancy or early childhood the resulting squint, if one developes, is in the great majority of cases convergent. A squint from the same cause in adult life is as often divergent as convergent, independent of the error of refraction. Convergence is a muscular effort and yet we find children with constant squints of 40° or more maintaining that amount of convergence all day and every day without any discomfort whatever and without any recognizable hypertrophy of the internal rectus. Is it possible for a person, child or adult, to maintain constantly a convergence of approximately 28 metre angles. If on the other hand the condition is due to esophoria, the eye is in a position of rest and needs no muscular effort to keep it so. This argument applies more forcibly to divergent squint, for in that case the patient maintains a divergence of 40° or more—a feat which we know to be impossible in the normal individual, but which is readily understandable if the condition be due to exophoria.

If we divide the squinting patients of my series into two age groups, one to ten and eleven to seventy-five, we find in the first group 589 convergent and 45 divergent squints or divergent to convergent 1 to 13·08 and in the second 145 convergent to 134 divergent or 1 divergent to 1·08 convergent. Comparing the same two age groups for heterophoria we find in the five to ten group exophoria to esophoria 1 to 2·45 and in the eleven to seventy-five group 1 to 1·06 or almost the same proportion as divergent to convergent squints. If we now take each year separately we find the proportion of exophoria to esophoria gradually increasing up to the age of eleven. In the sixth year there were twelve patients all with esophoria or 100%; in the seventh year twenty-one, fourteen with esophoria, five with exophoria and two with orthophoria or 66·6% with esophoria; in the eighth year there were thirty-two, twenty with esophoria, six with exophoria and six with orthophoria or 62·5%; in the ninth year there were twenty-three, fourteen with esophoria, six with exophoria and three with orthophoria or 60·8%; in the tenth year there were forty-two, twenty-three with esophoria, eleven with exophoria and eight with orthophoria or 54·7%; in

the eleventh year there were thirty-five, fifteen with esophoria, twelve with exophoria and eight with orthophoria or 42·7%. After the age of eleven the ratio becomes more uniform; twelfth year 36·1%, thirteenth year 36%, fourteenth year 51·6% and so on; the full figures are given in the table, in which it will be noticed that in only two years, the fourteenth and the twenty-fifth, does the percentage rise above fifty. I have not taken out the average after the fifty-first year as the numbers for each year separately are too small to be of value. The averages for the age group five to ten are esophoria 59·3%, exophoria 24·2% and orthophoria 16·3% and for the eleven to seventy-five group esophoria 33·4%, exophoria 31·2% and orthophoria 35·2%. The numbers are unfortunately too small to be conclusive, but they are large enough to be highly suggestive. If there were any method of estimating the heterophoria of infants, I believe that the percentage of esophoria would be even higher, but this is and I am afraid must remain merely speculation. The inference which I think may fairly be drawn from these figures, is that the great majority of children are born esophoric, that esophoria gradually diminishes up to the age of about eleven, that after that age the majority of people are either orthophoric or exophoric; according to these figures 33·4% esophoric to 66·4% exophoric and orthophoric. And there are reasons why the position of rest of the eyes should become stabilized at that age, for it is then that the globe attains its maximum growth and the axes of the orbit their maximum divergence.

Whitnall,[3] quoting Merkel, states that the nasomalar angle is 144° 6″ in children and 146° 5″ in adult females and 147° in adult males; so it is reasonable to suppose that the larger globes should find their position of rest in the direction of the axes of the orbit rather than converge in a direction at an angle to them.

If this be true, it will explain why a considerable proportion of convergent squints gradually become straight as the child grows older, for it is not the wearing of glasses which brings about this result, because they may become straight without glasses or any treatment whatever and the improvement is gradual with or without correction, whereas with glasses only that portion of the squint which is due to hypermetropia is corrected at once. It will also explain why convergent eyes which have been operated on by tenotomy in early childhood, sometimes become divergent in later life without any change in the refraction. A common explanation of this occurrence is that "the effect of the operation increases with time." This is certainly not correct; there is no evidence that the tendon of the internal rectus, once having become reattached to the globe, even shifts its position or that there is any progressive loss of power. There is even evidence that these things do not occur for the patient may have as ample an inward excursion of the globe as he ever had. I believe that such cases occur because the cause of the original squint was esophoria and the patient having ultimately become exophoric, is not in so advantageous a position to correct this as he would have been had the insertion of the internal rectus not been set back by the tenotomy. If such a child had not been operated on so early in life, the eyes would have become straight by themselves and no operation would have been needed. If tenotomy be done in older children, there is no subsequent divergence, at least I have not seen such a case, because the patient's position of rest of the globe has become stabilized, that is he has remained esophoric. Had he become exophoric or orthophoric, the eyes would have become straight by themselves. If he should become exceedingly exophoric, he would change from convergent to divergent even without any operation. Such cases are rare, but do occur. I think they are not commoner because the more pronounced cases of esophoria such as are the most likely to cause squint are the least likely to become extremely exophoric. Those cases in which, for some reason the internal rectus fails to become reattached to the globe, are in a different category. In such cases there is almost total loss of inward movement of the globe and the effect is practically immediate.

What part does myopia play in the causation of divergent squint? I venture to assert that as an error of refraction it plays no part at all, though indirectly through the lengthening of the globe it probably has a considerable effect, for one would naturally expect that the longer eye would find its position of rest in the longest axis of the orbit. As a matter of fact there is evidence that this is so, for Thomson[4] found that in the myopic type of children the ratio of exophoria to esophoria was 1 to 1·83 and in the hypermetropic type 1 to 4·69 or that exophoria was more than two and a half times as common in myopia as in hypermetropia. A divergent squint never corrects itself without operative interference, it is not affected by the correction of the error of refraction, it does not alter under an anæsthetic nor is it affected to any extent by tenotomy of the external rectus. Tenotomy does not cure exophoria or heterophoria of any kind for that matter, nor should we expect that it would; the globes are not pulled out of the parallel by the shortening or overaction of a muscle. They are in their position of rest without any one muscle being more exercised than any other. It is true that tenotomy will often relieve symptoms by rendering the correction of the defect easier and it may even correct the deformity of a squint by bringing the effort of correction within the reach of the desire for fusion. On the other hand, advancement of a muscle does cure heterophoria by rectifying actively the position of the globe in the orbit. Another argument against myopia being a cause of divergent squint is that a relaxation of accommodation cannot possibly cause an active divergence of the globes. It might possibly hinder a convergence, but cannot force the eyes past the parallel nor enable the patient to maintain a divergence of many degrees without discomfort. Further than that divergent squints are not associated with myopia in all cases. In my series there were 179 patients with divergent squint, in whom the refraction was known; of these one hundred were hypermetropic, fifty-six myopic and twenty-three emmetropic, or

55·8% hypermetropic, 31·2% myopic and 12·8% emmetropic, so that hyperopia was nearly twice as often associated with divergence as was myopia. If we divide these patients into the two age groups one to ten and eleven to seventy-five, there are in the first group forty-five patients with divergence, of which thirty-six are hypermetropic, four myopic and five emmetropic or 80% hypermetropic, 8·8% myopic and 11·1% emmetropic, hypermetropia being more than nine times as common as myopia. In the age group eleven to seventy-five there are one hundred and thirty-four divergent squints, of which sixty-four are hypermetropic, fifty-two myopic and eighteen emmetropic, or 47·7% hypermetropic, 38·8% myopic and 13·4% emmetropic. I think we may fairly conclude from these figures that myopia is not the commonest error of refraction in divergent squint. Nor is divergent squint the commonest form in myopia, at any rate in children. Ernest Thomson[5] found sixty-five squints in 790 patients with myopia in school children; of these twenty-nine were divergent and forty-five convergent or in other words convergent squint was one and a half times as common as divergent. I have no exact figures as to the incidence of squint in myopic adults, but myopia being defined as the condition in which any diameter in either eye is myopic, I found in ten thousand consecutive ametropic patients that myopia was in the ratio of 1 to 2·80 to hypermetropia; the ratio of divergent to convergent squint at all ages being 1 to 3·94, which is considerably lower, so that it is probable that divergent squint is not the commonest form in adult myopia. There is one fact from which one might argue that myopia is a causative factor in divergent squint, that is that myopia is much less common in convergent squint than is hypermetropia. In the 738 patients with convergent squint of my series only thirty-one were myopic, whilst 679 were hypermetropic, twenty-four emmetropic and in four the refraction was not known, but of these patients with convergent squint 589 were under the age of eleven years when at any rate in South Australia myopia is a very uncommon disease. It would seem then that myopia is more common in divergent than convergent squint, because both myopia and exophoria are conditions of adolescence and adult life and because myopic eyes have a greater tendency to become exophoric than hypermetropic ones.

In looking up the literature of concomitant squint I find that there are still some oculists who maintain that monocular amblyopia is the cause and not the effect of squint. There are several facts which to my mind provide conclusive evidence to the contrary, though I do not remember to have ever seen them categorically stated. They are: (i.) Squint may occur without amblyopia as in practically all truly alternating squints and in such divergent squints as develope after the age of six years; (ii.) amblyopia may occur without squint as in cases of disease or injury to one eye and of marked anisometropia (the cause in this latter case is probably the same as in squint, the brain having suppressed the blurred image of the more ametropic eye, finally becomes incapable of seeing it at all); (iii.) amblyopia may be cured if suitable treatment be applied at an early enough stage; (iv.) Worth records cases in which through prolonged occlusion of the fixing eye that eye has become the squinting amblyopic one and the previously squinting one has become the fixing and seeing eye; (v.) the amblyopic eye is practically always that which is most ametropic, not necessarily greatly so, for a difference of 0·5 diopter of astigmatism may be enough to determine which shall be the fixing and which the deviating eye. It is not contended by anyone that the error of refraction is the cause of the amblyopia, therefore it seems to be too remarkable a coincidence or rather series of coincidences that the amblyopia should always occur in that eye, however slight the difference may be. (vi.) The amblyopia may persist although the squint has rectified itself. It is quite common to find people with eyes perfectly aligned, but with one amblyopic, who, on being questioned, tell us that they formerly had a squint. It is difficult to understand how the effect can disappear while the cause remains. At the same time one must concede that a congenital monocular amblyopia, if there be such a thing, would be a potent factor in the causation of squint and it is, of course, possible that some squints may be of this kind. I am using the term amblyopia for such patients as have a defect of vision without any physical defect to account for it. Congenital amblyopia is common enough from physical causes such as lamellar cataract, albinism and coloboma of the chorioid, but in these cases both eyes are affected and there is usually no squint.

A more difficult question to decide is whether the absence or weakness of the fusion faculty is congenital or acquired as the result of the squint. There seems to be no reason why it should not be lost just as is the sight in the amblyopic eye as a result of the repeated suppression of the second image. Worth and others have had success in bringing the fusion sense to normal by the use of his amblyoscope, just as the sight is regained in the amblyopic eye by suitable training; on the other hand if the condition be never congenital, it would be difficult to explain those divergent squints which develope after the sixth year in which amblyopia does not occur and in which one would expect the fusion sense to be well developed had it ever been present. Certainly a permanent squint is never present without a weak fusion sense, though an occasional or latent one may be. A latent squint differs only from a constant one in that the fusion faculty is well developed. Should binocular vision be lost in such a case through blindness of one eye, the squint at once becomes constant. On the other hand, it is not uncommon to find an almost total absence of the fusion sense without any squint at all, nor is there any reason why absence of binocular vision should cause a squint in the absence of any contributing factor. Worth's essentially alternating squint which he attributes entirely to absence of fusion sense, differs only from his accidental alternating squint in that the whole of the deviation is due to heterophoria; otherwise they are exactly the

same. The question as to whether a squint shall be alternating or constant is settled by the equality or inequality of the refraction, whether this be ametropic or emmetropic. That the fusion sense is not equally developed in all people is shown by the fact that although most of us are able to use the ophthalmoscope or the microscope with both eyes open there are some people who are never able to suppress the second image and have to close one eye; also by the varying degree and length of time during which patients with a permanent paralysis of an ocular muscle are troubled by the diplopia.

To sum up there are three causes of concomitant squint. These are in the order of their importance: (i.) Weakness of the fusion sense, (ii.) heterophoria, (iii.) hypermetropia. The first only is essential, but must be accompanied by one of the others. The determining factor in the development of a squint is the relation between the desire for fusion and the effort needed to align the eyes correctly. If the desire for binocular vision be greater than the effort, there will be no squint. If the effort be greater than the desire, then a squint will result.

Age.	Number of Patients.	Relative Percentage of Heterophoria in—		
		Esophoric Eyes.	Exophoric Eyes.	Orthophoric Eyes.
5	12	100	0	0
6	21	66·6	23·8	9·5
7	32	62·5	18·7	18·7
8	23	60·8	26·0	13·0
9	42	54·7	26·1	19·0
10	35	42·8	34·2	22·8
11	36	36·1	27·7	36·1
12	23	34·7	21·7	43·4
13	31	51·6	16·1	32·2
14	24	41·6	33·3	25·0
15	20	40·0	20·0	40·0
16	22	40·9	31·8	27·2
17	28	42·8	21·4	35·7
18	34	35·2	26·4	38·2
19	33	27·2	27·2	45·4
20	40	32·5	32·5	35·0
21	43	23·2	46·5	30·2
22	26	46·1	34·6	19·2
23	44	34·0	40·9	25·0
24	32	56·2	15·6	28·1
25	33	27·2	27·2	45·4
26	33	36·3	27·2	36·3
27	37	32·4	24·3	43·2
28	35	28·5	37·1	34·2
29	37	43·2	35·1	21·6
30	38	28·9	50·0	21·0
31	34	26·4	26·4	47·1
32	41	34·1	24·3	41·4
33	39	28·2	30·7	41·0
34	32	15·6	37·5	46·8
35	42	35·7	30·9	33·3
36	34	26·4	35·2	38·2
37	33	48·4	24·2	27·2
38	41	41·4	31·7	26·8
39	40	37·5	15·0	47·5
40	41	36·5	34·1	29·2
41	23	34·7	43·2	21·7
42	40	27·5	35·0	37·5
43	36	36·1	30·5	33·3
44	39	20·5	35·8	43·5
45	38	18·4	36·8	44·6
46	30	36·6	16·6	46·6
47	29	27·5	24·1	48·2
48	29	48·2	27·5	24·1
49	35	25·7	31·4	42·8
50	37	45·9	27·0	27·0

All ages from 5 to 75 (1,776 patients): Esophoria, 35·8%; exophoria, 30·7%; orthophoria, 33·5%.
All ages from 5 to 10 (165 patients): Esophoria, 59·3%; exophoria, 24·2%; orthophoria, 16·3%.
All ages from 11 to 75 (1,601 patients): Esophoria, 33·4%; exophoria, 31·2%; orthophoria, 35·2%.
All ages: Exophoria is to esophoria as 1 : 1·16.
5 to 10: Exophoria is to esophoria as 1 : 2·45.
11 to 75: Exophoria is to esophoria as 1 : 1·06.

Hypermetropia plays a very minor part in the causation of convergent squint. Myopia as an error of refraction has no part in the causation of divergent squint, though it may have indirectly owing to the greater tendency to exophoria in myopic eyes.

Age.	Esophoria.	Exophoria.	Orthophoria.
5	12	0	0
6	14	5	2
7	20	6	6
8	14	6	3
9	23	11	8
10	15	12	8
11	13	10	13
12	8	5	10
13	16	5	10
14	10	8	6
15	8	4	8
16	9	7	6
17	12	6	10
18	12	9	13
19	9	9	15
20	13	13	14
21	10	20	13
22	12	9	5
23	15	18	11
24	18	5	9
25	9	9	15
26	12	9	12
27	12	9	16
28	10	13	12
29	16	13	8
30	11	19	8
31	9	9	16
32	14	10	17
33	11	12	16
34	5	12	15
35	15	13	14
36	9	12	13
37	16	8	9
38	17	13	11
39	15	6	19
40	15	14	12
41	8	10	5
42	11	14	15
43	13	11	12
44	8	14	17
45	7	14	17
46	11	5	14
47	8	7	14
48	14	8	7
49	9	11	15
50	17	10	10
51	7	4	6
52	7	7	2
53	5	8	5
54	6	5	5
55	2	10	5
56	5	8	12
57	5	4	1
58	5	2	9
59	3	5	2
60	3	7	6
61	3	0	2
62	1	5	6
63	0	4	1
64	3	4	5
65	1	2	5
66	0	0	1
67	1	4	0
68	3	3	4
69	0	2	2
70	2	2	2
71	2	1	0
72	0	0	1
73	2	1	0
74	2	0	1
75	0	2	0

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(1) Claud Worth: "Squint: Its Causes, Pathology and Treatment."
(2) F. W. Marlow: "The Relative Position of Rest of the Eyes and the Prolonged Occlusion Test."
(3) S. Ernest Whitnall: "Anatomy of the Human Orbit."
(4) Ernest Thomson: "A Note on the Lateral Ocular Muscle Balance in School Children with Special Reference to Squint in Myopia," *The British Journal of Ophthalmology*, Vol. IX., No. 3.
(5) Ernest Thomson: "Some Statistics of Myopia in School Children with Remarks Thereon," *The British Journal of Ophthalmology*, Vol. III., No. 7.

SIR JAMES BARRETT (Melbourne) congratulated the speaker on the amount of work he had put into his paper. He quoted the work of Lang and himself in disproving the old idea that amblyopia was the cause of squint. He had examined numbers of patients and he found that amblyopia had nothing to do with it. While there seemed to be many anomalous cases his experience was that the majority were due to hypermetropia and mentioned the condition in which squint disappeared with suitable glasses and immediately reappeared when glasses were discarded. He had seen convergent non-paralytic squint following the use of forceps. He agreed with the speaker that squints occasionally disappeared without any treatment and said that operations on that account used to be postponed till the age of fifteen or sixteen.

DR. F. ANTILL POCKLEY (Sydney) agreed with the previous speaker that hypermeteropia was most often associated with convergent squint and mentioned the fact that if a mydriatic was used in the fixing eye, the other eye squinted.

DR. G. W. HARTY (Wellington) mentioned reflex causes which appeared to cause squint, for example thread worms. He had treated patients in whom squint disappeared after thread worms had been removed. He believed in having the faulty eye trained daily for short periods at regular intervals with the use of letters, pictures and the like and thought it was the first duty to encourage the retention of acuity by training as well as glasses. He thought that this was important whether the person got binocular vision or not.

DR. GEORGE FENWICK (Auckland) congratulated Dr. Morgan on his work and pointed out that early operation by placing the muscles in the most advantageous anatomical position should result in the preservation of binocular visien in a satisfactory percentage of patients. He asked whether this had been the President's experience.

DR. H. F. SHORNEY (Adelaide) asked Dr. Morgan to explain the mechanism of alternating squint according to his theory.

DR. F. M. MORGAN in reply said that he did not operate before the age of fifteen and in reply to Dr. Harty with regard to the temporary causation of squint by thread worms said that if a child was run down or debilitated he squinted.

In answer to a question of Sir James Barrett he said that glasses corrected only a certain amount of the squint.

THE OCULAR SIGNS OF EXOPHTHALMIC GOÏTRE.

By J. BROOK LEWIS, M.B., B.S. (Melbourne),
Honorary Assistant Ophthalmologist, Adelaide Hospital.

THE ocular signs of exophthalmic goïtre are many, but a patient may come under notice in which they are all absent.

The only sign, ocular or general, which is constant in exophthalmic goïtre, is tachycardia. For convenience of description the signs may be grouped as follows: (i) Exophthalmos, the ætiology of which is still undetermined, (ii) signs due to alterations in the relation of the eyeball muscles, (iii) signs due to changes in the metabolism of the eye, (iv) signs due to vascular alterations in the orbit and adnexa, (v) anomalies of lachrymation, (vi) signs resulting from exposure of the front of the eyeball.

EXOPHTHALMOS.

Exophthalmos is absent in 20% of patients with exophthalmic goïtre (Roemer).

In the earlier stages of the disease the exophthalmos may be only slight; this increases as time goes on and the usual appearance of "petrified fright" becomes more marked. In some cases the exopthalmos is marked from the beginning of the case.

The eyes are usually directed straight forwards in the line of the optical axes, but the direction may be lateral or oblique.

The amount of exophthalmos is the same in both eyes, but may be more evident in one eye or altogether absent on one side. Hill Griffith found unilateral exophthalmos in 22% of cases and Willbrand and Saenger in four out of twenty-seven cases.

The exophthalmos at first unilateral may later on become bilateral.

The amount of exophthalmos may vary at different times during the course of the disease and becomes increased during excitement, exertion, active attention and menstruation.[1]

In unilateral exophthalmos the thyreoid is usually more enlarged on the side corresponding to the exophthalmos, though the thyreoid may be enlarged on one side and a unilateral exophthalmos be present on the other.

Sometimes in the presence of exophthalmos the goïtre may be entirely absent.

The exophthalmic eye can be pressed back by the hand to a slight degree, but returns when the pressure is released.

Stooping or pressure on the jugular vein increases the exophthalmos in a slight degree.

Sattler has shown that pressure on the facial veins with the head upright increases decidedly the amount of exophthalmos. The exophthalmos may be so great that the lids are unable to cover the eyes and the eyes may be dislocated outside the lids, a condition that may recur after reduction.

The exophthalmos is one of the last symptoms of exophthalmic goïtre to disappear and may persist for years. In one case it persisted for forty-nine years (Goulden, quoted by Foster Moore) and in a case of my own seventeen years after the other symptoms had subsided.

One eye may improve more rapidly than the other.

After thyreoidectomy the eye on the side operated may subside and the other remain exophthalmic.

Ætiology of the Exophthalmos.

It is evident that a case of exophthalmos which is sudden in onset, within a few hours, must have a different ætiology to one in which the exophthalmos takes months to develope.

As regards the acute onset of the disease the following cases are quoted.

Lauder Brunton[2] tells of a case in which the patient was greatly agitated by a thunder storm and in thirteen hours or less the eyes had become protuberant. In this case the disease became chronic.

Solbrig (quoted by Brunton) reports a case where the disease became fully developed in a day, but disappeared in ten days.

Osler quotes a case in which the symptoms came on with great rapidity and the patient died on the third day.

Trousseau tells of a woman in whom the symptoms of the disease developed in a single night

which she had spent in tears lamenting her father's death.

Foster Moore, on the other hand, is inclined to think judging from his own experiences and that of the physicians at Saint Bartholomew's Hospital, that sudden exophthalmos does not occur.

In the comparatively few examinations made after death the only condition found in the orbits was an increase in the orbital fat.

If we are to accept a sudden onset in some cases it is necessary to accept an overfilling of the orbit with fluid (either intra- or extravascular) which it is possible to ascribe to a disturbance, as in the case of the muscles, in the sympathetic nerve supply.

Bram in his book[1] summarizes the ætiology of exophthalmic goître as follows:

(i) A deposit of fat behind the eyeball.

(ii) Continuous contraction of the *levator palpebræ* muscle causing a contraction of the lids and exposure of the sclera.

(iii) Venous congestion of the posterior part of the orbit. J. C. O'Day believes that the exophthalmos is due to stasis in the ophthalmic veins due in turn to tetany of the ventricles.

(iv) Dilatation of the retrobulbar arteries.

(v) Contraction of Müller's muscle. This latter consists of a small group of muscular fibres located between the sphenomaxillary fissure and the supra-orbital groove.

(vi) Landström (quoted by C. H. Mayo) describes a microscopical muscle occurring in streaked layers in the fascia behind the eyeball and about the globe. This muscular sheath is attached forward to the lids and anterior orbital fascia. It is controlled by the sympathetic nervous system and serves to oppose the four orbital muscles which draw the eyeball back. Irritation of Landström's muscles directly or through the cervical sympathetic causes exophthalmos and the concomitant drawing backward of the lids. This phenomenon is produced with the assistance of Müller's muscle.

(vii) Maurice believes that the exophthalmos is probably due to an excessive action of the adrenals.[3]

(viii) Marine and Lenhart, however, conclude that in view of the fact that exophthalmos may occur without thyreoid hyperplasia and that there may be considerable thyreoid hyperplasia without exophthalmos, both phenomena through often not synchronous are manifestations of a fundamental and more obscure nutritional disturbance. These authorities believe that the thyreoid hyperplasia is not a direct ætiological cause of exophthalmos.[4]

Ocular Signs in Exophthalmic Goître due to Alterations in the Relations of the Eye Muscles.

Von Graefe's Sign.

When a patient with exophthalmic goître is asked to look down, the ball of the eye descends as in a normal person, but the upper lid which in health descends concomitantly with the ball, either executes this descending movement slowly and jerkily or in some cases not at all, leaving a portion of the white of the eye below the edge of the upper lid.

This is known as von Graefe's sign and is one of the classical signs of exophthalmic goître. As a modification of this sign Solomon Solis Cohen quoted by Bram states that in some cases the eyelid in its downward course stops short (the hitch) and then goes further down to conclude its descent imperfectly; when the lid is raised there is again a hitch before the movement is completed. Solomon Solis Cohen calls this "hitch" sign the larval stage of von Graefe's sign.

Von Graefe's sign is not always present in exophthalmic goître. According to Posey and Spiller[5] out of 311 cases collected from several authors it occurred ninety-three times. In seventeen cases brought under my notice it occurred seven times.

Von Graefe's sign has been observed in healthy individuals and also in a number of diseases. Sharkey[6] observed it twelve times in 613 cases of all forms of disease including exophthalmic goître. It has been observed in nervous diseases and pareses of the ocular muscles.

Kocher and Jessup have produced in a modified form von Graefe's sign by putting cocaine into the eye and considered it to be due to a direct excitation of Müller's muscle through the sympathetic.

An imperfect movement of the lower lid has also been noted, but it is not so definite as in the upper lid owing to the greater movement of the globe in a downward direction.

Von Graefe's sign may appear before the exophthalmos and persist after its subsidence and may come and go while the disease is in progress, which may account for the wide variation in frequency quoted by several authors (from 8% to 50%).

Von Graefe's sign has been observed in one eye alone. In unilateral exophthalmos the lid symptoms are only present on the affected side.

Von Graefe's sign is sometimes met with when all other signs of exophthalmic goître are absent.

Stellwag's Sign.

Stellwag's sign consists in a diminution in the number of winks and at the same time the action is imperfectly performed.

Roemer has written: "While we normally wink five to ten times a minute at unequal intervals, a patient with exophthalmic goître scarcely winks once a minute."

Sometimes the winks are frequent for a short time and then there are periods of rest.

The lids do not come together and so the action is incomplete.

This symptom may occur only in one eye and it is increased by fatigue, excitement or mental anxiety.

Sometimes the opposite condition prevails and the eyes wink too frequently.

It is a prominent symptom in exophthalmic goître and its presence causes the tardy removal of foreign bodies lodged on the cornea from which ulceration may result.

Moebius's Sign.

Moebius's sign consists in a lessening or absence of convergence and is independent of the difficulty of converging the enlarged globes.

The insufficiency of convergence causes asthenopia, but not diplopia.

Dalrymple's Sign.

Dalrymple's sign consists in a widening of the palpebral fissure, so that the white of the sclerotic is seen above and below the cornea.

This gives the patient a staring look as if he were experiencing astonishment and fear. A similar effect is produced by cocaine.

This sign is not due to the exophthalmos, as it has been seen when the exophthalmos had not developed.

The widening of the palpebral fissure may, however, increase the exophthalmos.

Dalrymple's sign may occur in one eye only. During sleep the lids are perfectly closed except when the exophthalmos is considerable.

Gifford's Sign.

It is well nigh impossible in the subject of exophthalmic goître to evert the upper lid and when this is accomplished the tension is so great that the lid is quite white.

Gifford's sign is not by any means constant in the fully developed malady, although present in the early stages of the disease.

It is due to the upper lid remaining behind when the patient looks down aided in some cases by the swelling and œdema of the subcutaneous tissues which is present in exophthalmic goître.

Suker's Sign.

Suker (quoted by Zentmayer[7]) described an ocular symptom of hyperthyreoidism.

After extreme lateral rotation of the eyes to the right or to the left with head fixed and fixation maintained for a second or two on attempting to follow the fixation point as it is rapidly swung into the median line one or other of the eyes fails to follow in a complementary manner into proper convergence for this point when it is brought into the median line.

Either the right or left eye makes a sudden rotation into fixation with its fellow, but before it does so an apparent strabismus is manifest.

Suker believes this phenomenon to be due to a dissociation in the functions of the sympathetic and the extraocular motor nerves of the eyes and perhaps also to exhaustion on extreme lateral rotation of the eyes.

Kocher's Sign.

If a patient is asked to view an object moving in a vertical direction, the upper lid moves suddenly upward leaving the eyeball behind.

Pareses of the Ocular Muscles.

Pareses of the ocular muscles may be met with in grave cases of this disease, especially in the form of bilateral *ophthalmoplegia externa*. Pareses of associated muscles have also been observed.

Pareses of individual muscles occur, but they are rather rare.

In severe exophthalmos there is frequently more or less restriction of the movements of the eyeball, although, as the limitation is the same in both eyes, the patient suffers little.

Tremor of the Eyelids.

Tremor may be noted in the upper lids when the eyelids are gently closed. It is characterized by its rapid appearance when the lids are closed, by its regularity and rhythm and its very fine character.

Nystagmus.

Nystagmus occurring in patients with exophthalmic goître has been reported. Freund believes it to be similar to the tremor occurring in the extremities.

Reversed Argyll-Robertson Pupil.

Curschman (quoted by McCarrison) notes that in many instances the eyes present in the phenomenon just the reverse of the Argyll-Robertson pupil, that is there is reaction to light, but not to convergence.

Mydriasis due to Adrenalin.

Curschman and Loewi have observed that in a considerable proportion of patients with Graves's disease the installation of adrenalin results in mydriasis.[8]

SIGNS DUE TO ALTERATIONS IN THE METABOLISM OF THE EYE.

Cataract.

Kirkpatrick has written: "Vossius attempted to show an association between cataract and goître, but failed to establish the connexion; this, however, is hardly to be wondered at since the activity of the thyreoid and possibly of the parathyreoids is a variable quantity in goître."

Optic Atrophy, Neuritis and Amblyopia.

Roemer says: "Coppez has shown that visual disturbances may arise after the prolonged excessive administration of thyreoid."

Birsch-Hirschfeld as well as Nobius has been able to produce in dogs an atrophy of the optic nerve with pronounced symptoms of degeneration in the retina by long overfeeding with thyroidin.

Although vascular changes in the retina and in some cases optic neuritis, retrobulbar neuritis and optic atrophy have been recorded, they are very rare and should be regarded as complications.

Falling out of the Eyelashes and Eyebrows.

Falling out of the eyelashes and eyebrows has been frequently recorded and is a part of the alopecia which is often seen in hyperthyreoidism.

The Jellinck-Teillais Sign.

In the Jellinck-Teillais sign a brownish discoloration of the eyelids is found around the orbital

margin. It varies in colour with a violet to a deep brown and occurs in 10% to 25% of all patients with hyperthyreoidism.

Sainton and Fayolle state that it is often a feature of the earliest stage of the disease, later almost disappearing with subsequent recurrence.

Ocular Signs due to Vascular Alterations in the Orbit and its Adnexa.

Bruit Over the Eyeball.

Many observers have recorded a bruit heard when the stethoscope is placed over the closed eyelids.

Some observers have ascribed it to muscular contraction of the eyelids. It is found also in aortic regurgitation.

Œdema of the Eyelids.

Œdema of the eyelids occurs either in an early or late stage of the disease and is often present after the other signs have subsided.

Obliteration of the Superior Sulcus Orbito-Palpebralis.

The amount of fluid in the orbit is increased in all cases and in some may cause the obliteration of the *sulcus orbito-palpebralis.*

Anomalies of Lachrymation.

Berger states that epiphora may occur before Stellwag's and Dalrymple's signs are present and is present at night when the secretion of tears ceases. He ascribes it to an increased activity of the sympathetic vasomotor fibres.

Other authorities put it down to two causes: (i) the conjunctiva being exposed and thus more subject to irritation by the air, (ii) owing to the imperfect performance of winking the tears are not drawn into the lachrymal canaliculi.

Dryness of the cornea occurs in patients who have had the disease for a long time and is due to the lessened sensibility of the cornea to ordinary excitants, as the air and dust, and also because the large surface of conjunctiva exposed accounts for the rapid evaporation of tears.

Ocular Signs due to Exposure of the Eye.

Conjunctivitis.

Owing to the exposure of the cornea and sclerotic in exophthalmic goître and the imperfect performance of winking chronic conjunctivitis is a not infrequent accompaniment.

Keratitis.

Several factors tend to make these patients liable to keratitis:

(i) The exophthalmos and consequent exposure of the cornea to irritants, (ii) the imperfect performance and the infrequency of winking leading to the deposit and non-removal of various irritants to which the eye is exposed, (iii) the retraction of the upper lid which helps to expose the cornea and sclerotic more effectively, (iv) Sattler says that toxic substances circulating in the blood affect injuriously the nutrition of the cornea.

The keratitis may vary from a slight haziness and lack of lustre in the cornea through superficial infiltrations to ulceration. The cornea may become ulcerated in a very short time.

It is remarkable how well a cornea which is affected with keratitis, may recover after the performance of tarsorrhaphy.

Panophthalmitis.

The keratitis, if not treated or in some severe cases even with treatment, may go on to suppuration and perforation of the globe with resultant panophthalmitis.

References.

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(2) T. Lauder Brunton: Gibbon's "Medicine" (1901).

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(4) Marine and Lenhart: *Journal of Experimental Medicine*, 1910, page 311.

(5) Posey and Spiller: "The Eye and the Nervous System."

(6) S. J. Sharkey: *The Lancet*, 1890, page 877.

(7) Zentmayer: "The Eye and the Endocrine Organs." *The Journal of the American Medical Association*, July 7, 1917, page 1.

(8) Curschman and Loewi: *Zeitschrift für Klinische Medizin*, 1912.

Dr. A. M. Morgan (Adelaide) said that patients with exophthalmos very rarely came under his notice. The patients sought the aid of general surgeons.

Sir James Barrett (Melbourne) said that one of his patients had taken thyreoid extract to reduce his weight. He had brought it down 12·6 kilograms (two stone), but had developed exophthalmos.

Dr. A. J. Hall (Dunedin) quoted the case of a woman who had heard of the sudden death of a child from scarlet fever and had developed double exophthalmos. He did so in view of the fact that Foster Moore who had done a great deal of work in this connexion, did not mention any cases of sudden onset.

THE OPERATIVE TREATMENT OF LACHRYMAL OBSTRUCTION.

By E. L. Marchant, M.B., B.S. (New Zealand), *Wellington.*

Some explanation is necessary as to why it has fallen to me to open this paper. Dr. Kent Hughes was to have done so but was unable to come over. Some apology is due also on account of its brevity, but very short notice was given and rather than have this interesting and important subject left out, I agreed to open the discussion and hope that those with much wider experience than mine will give their views and that fruitful discussion may ensue.

There is no necessity to discuss the ordinary treatment of lachrymal obstruction, as this is presumed to have failed and operation been decided upon. The intranasal operation of West and others leaves much to be desired, as in some conformations of the nose it is difficult to be sure exactly how large an opening has been made in the sac wall. A probe may pass very freely and yet sufficient tissue may not have been removed for a satisfactory result.

In one case of my own the passage was full and fluid could be easily syringed through, but functionally it was a failure. This was a case of congenital obstruction of nineteen years' duration without sepsis and the sac was very small. It is particularly in this type of case that the intranasal operation is liable to fail, whereas those with dilated sacs give good results, although recurrence of epiphora may take place months after what was originally a successful operation. In casting about for a better operation I found that the Mosher-Toti appeared to be a reasonably radical procedure free from the difficulties of access sometimes encountered in that of West and offering a much wider passage from the canaliculus to the nasal cavity.

The classical incision is given as lying six millimetres from the inner canthus of the eye, starting opposite the crease of the upper lid and running downwards in a nearly straight line parallel to the lachrymal groove to a point three millimetres below the level of the lower orbital margin. Perfect anæsthesia can be obtained by "Novocain" injection and cocaine packing in the nasal cavity. Even if general anæsthesia is to be used great resistance is given by skin injection of "Novocain" and adrenalin since hæmorrhage is usually free. The incision is made down to the periosteum, the sac identified and turned outwards from the groove in its entire length, until the commencement of the nasal duct is seen. The floor of the groove is then broken through into the nasal cavity and the opening enlarged by cutting away portion of the posterior margin of the ascending process of the superior maxilla with a Citelli's punch forceps and the rest of the lachrymal bone. In the first double case operated upon by myself, failure occurred on one side and the operation was repeated, when the blockage was found to be due to insufficient removal of this posterior margin of the ascending process with the result that the opening of the sac was resting against the cut bony surface. Freer removal of bone allowed perfect drainage.

An essential of this operation is to lay open not only the inner sac wall, but also that of the entire length of the nasal duct. Having some difficulty in being sure exactly how much of the lumen of the sac had really been removed, I found that an easy and certain method was to tent in the inner sac wall by a probe through the canaliculus and then to transfix the sac by a straight needle into the nasal cavity. If then the sac be divided vertically external to the needle, the whole lumen must be included. Care must be taken not to remove so much as to damage the common opening of the canaliculi or the result will be ruined.

The opening up of the nasal duct is advised to be done by a Kerison's punch, but being unable to obtain this at the time, I found an easy method was to pass a No. 1 slightly bent knitting pin down the duct, place a small gouge external to it and drive this down the duct, using the pin as a guide. The whole inner wall is thus ripped open and can then be easily and completely removed by any small conchotome. All mucous membrane tags and bone spicules must be carefully removed from the operation field in the nose.

The success of the operation depends on making large holes and insuring that the opening in the sac coincides with that in the bone.

After the wound has been sutured, a pad and bandage are advisable for twenty-four hours to prevent extravasation of blood into the tissues. After that period painting with Friar's balsam is sufficient.

After treatment consists of syringing only once or twice and attending to any possible overgrowth of granulations from the cut bony surface in the nose.

It is not insisted that the above operation is the best, as there are simplifications untried by myself of which others may tell us nor is it certain that the operation will render the conjunctival sac sterile to allow of an intraocular operation.

What appears to be a simple operation described as fool-proof is reported by F. H. Hanger in Volume XXXV of *The Laryngoscope*. A No. 3 Theobald's probe is passed from the lower punctum down the nasal duct and its lower end located by detaching the anterior end of the inferior turbinate. The probe is then withdrawn and replaced by the author's probe pointed cannula loaded with a length of No. 3 ligature silk, whose lower end is then withdrawn from the anterior nares and the cannula removed. The author's special punch forceps have a hole in the tip of the male blade and this is securely tied on to the lower end of the silk. On pulling the upper end of the silk the male blade engages the nasal duct whose inner wall and that of the lachrymal sac are punched out as the forceps are drawn upwards. Hanger reports only one failure in sixteen cases, but I presume that either only patients in whom a probe could be passed, were selected or else that the making of a false passage does not interfere with the result.

A further operation described by Poyales, of Madrid, appears to aim at a very wide opening from the inner canthus into the nasal cavity. This is done by cutting longitudinal flaps from the conjunctival surfaces of the canaliculi, excising the lachrymal sac, opening the lachrymal groove into the nasal cavity and turning in the flaps. The canaliculi thus laid open form a trench directly into the nasal cavity. The operation sounds somewhat difficult, but would apparently give perfect drainage and might prove satisfactory when there was stricture of a canaliculus.

DR. A. M. MORGAN (Adelaide) said that he favoured operation for removal of the lachrymal sac in cases of nasal obstruction and was quite satisfied with the results he got from this operation.

DR. G. W. HARTY (Wellington) said that he had had good results from the operation and that recurrence took place when it was difficult to get good drainage. He agreed with the speaker that a large opening was necessary.

DR. L. S. TALBOT (Timaru) said he had operated on several of these patients but had found that after some time there was a tendency to cicatricial contraction. He would ask if it was sufficient to provide drainage only; if so, it was almost like going back to the cannular stage.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. H. TEBBUTT.

FIGURE I. — Lymphadenoid Goitre.

A—Thickened capsule blood vessel; B—Collection of small acini with small colloid droplets; C—Lymphocytic aggregation obliterating a gland unit; D—Lymph sinusoid; E—Small acini; F—Diffuse lymphocytic infiltration with a few atrophic gland acini; G—Lymphocytes.

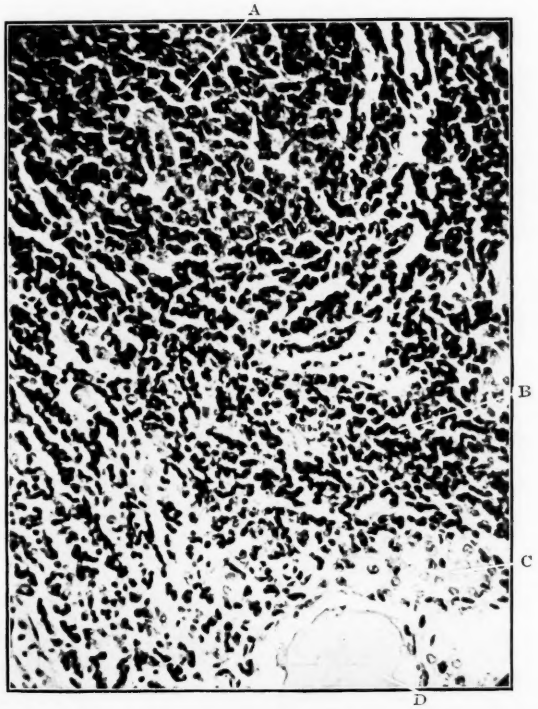

FIGURE II.—Lymphadenoid Goitre, probably early lesion.

A—Germ centre; B—Diffuse infiltration by lymphocytes and plasma cells; C—Two small acini becoming involved in the infiltration; D—Large alveolus lined by flat epithelium and containing colloid.

FIGURE III.—Lymphadenoid Goitre, later stage than Figure II (intermediate stage).

A—Diffuse lymphocytic infiltration and plasma cells; B—Syncytial mass; C—Fibrous tissue; D—Syncytial mass; E—Groups of thyreoid cells tending to form syncytial masses; F—Thick fibrous trabeculum.

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ILLUSTRATIONS TO THE ARTICLE BY DR. A. H. TEBBUTT.

FIGURE IV.—Lymphadenoid Goitre, strong diffuse infiltration with lymphocytes and plasma cells (intermediate stage).
A—Thyreoid cells; B—Fibrous tissue band; C—Thyreoid cells; D—Syncytial mass.

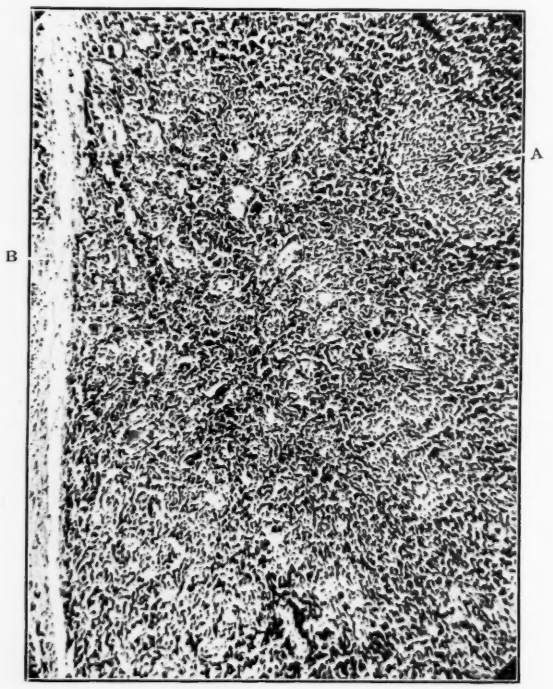

FIGURE V.—Lymphadenoid Goitre (intermediate stage).
A—Germ centre. Scattered through the diffuse infiltration are minute acini containing a little colloid (clear areas) and also syncytial masses of small size. See next photograph. B—Trabecula of fibrous tissue.

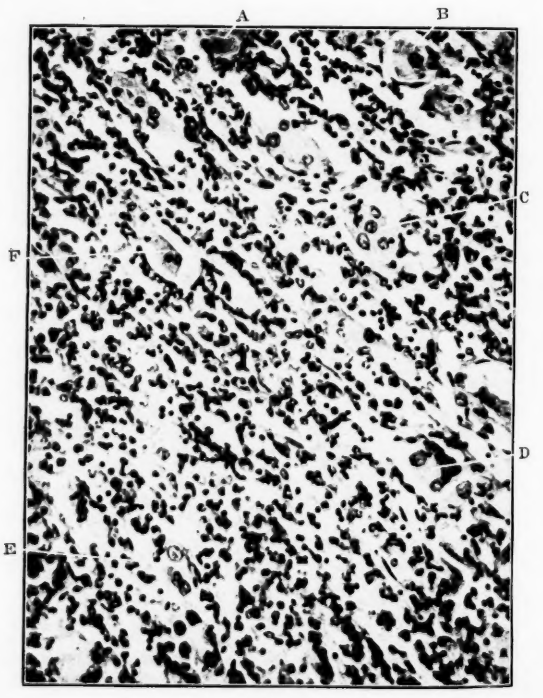

FIGURE VI.—Lymphadenoid Goitre (intermediate stage).
A—Droplet of colloid in a minute acinus; B—Thryeoid cell or cells; C, D, E—Syncytial mass, note the diffuse infiltration with lymphocytes and plasma cells; F—Large thyreoid cell or two fused cells.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. H. TEBBUTT.

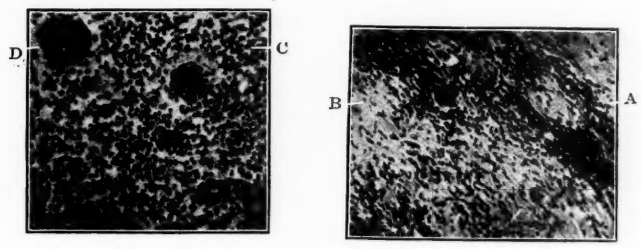

FIGURE VII.—Advanced fibrous atrophy with groups and streaks of round cells. A—Round cells (mainly lymphocytes); B—Lymphocytes (and plasma cells); C—Groups of epithelial cells; D—Fibrous tissue.

(From Virchow's Archiv für pathologische Anatomie und Physiologie.)

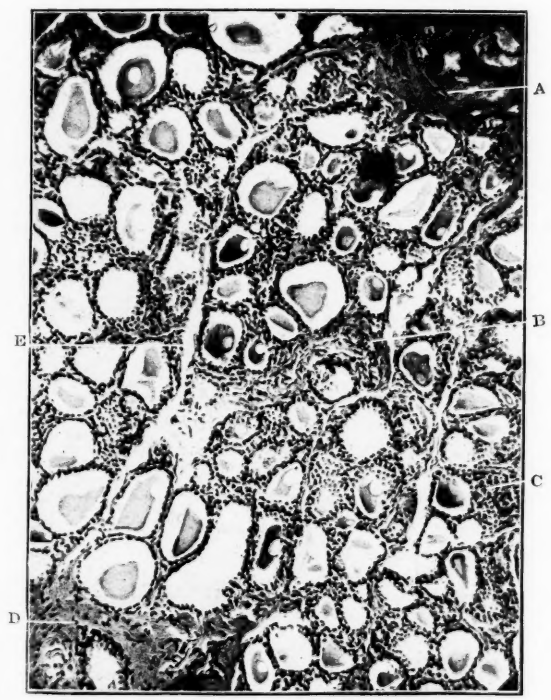

FIGURE VIII.—Thyreoid of an infant with increase of fibrous tissue.

A—Fibrous knot; B—Fibrosis; C—Interalveolar cells; D—Fibrosis; E—Lymph sinus.

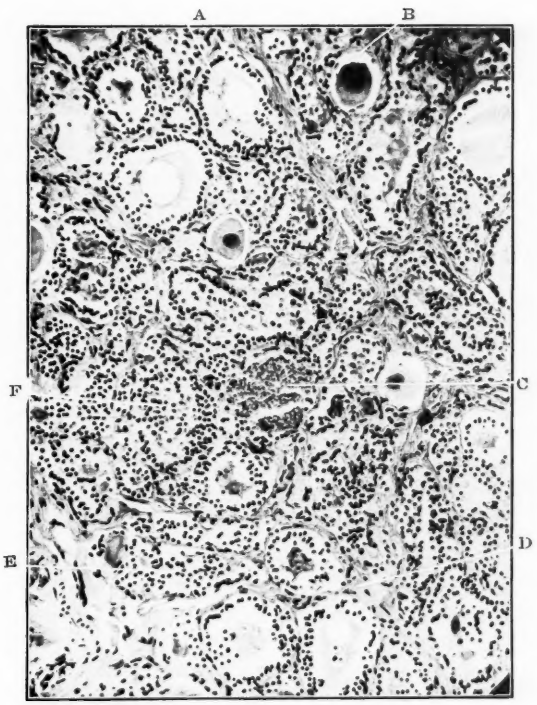

FIGURE IX.—Thyreoid of man aged 42, with intra-lobular fibrosis.

A—Colloid acinus lined by columnar epithelium; B—Alveolus containing deeply stained colloid and atrophied epithelium; C—Alveolus filled with globules. Altered red blood cells; D—Intralobular fibrosis separating alveoli; E—Groups of small noncolloid alveoli; F—Alveolus showing catarrh.

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ILLUSTRATIONS TO THE ARTICLE BY DR. A. H. TEBBUTT.

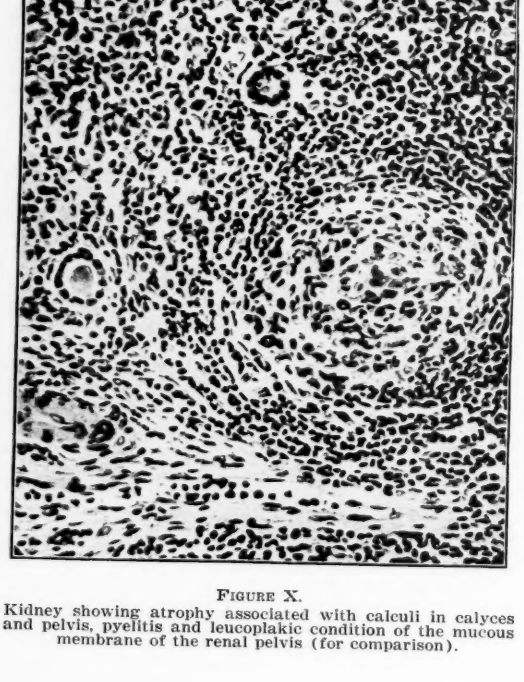

FIGURE X.
Kidney showing atrophy associated with calculi in calyces and pelvis, pyelitis and leucoplakic condition of the mucous membrane of the renal pelvis (for comparison).

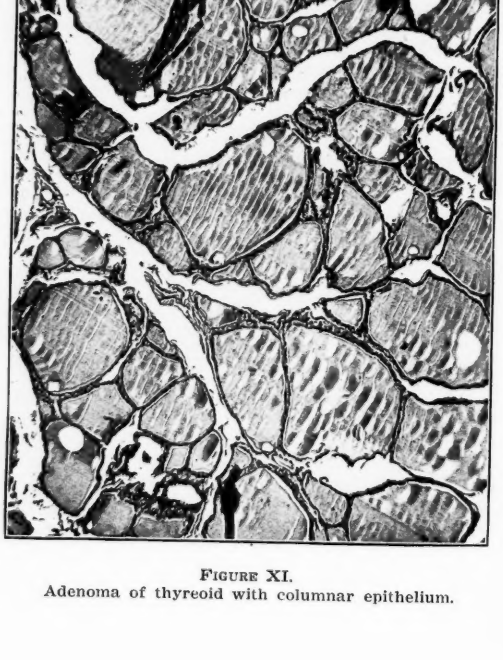

FIGURE XI.
Adenoma of thyreoid with columnar epithelium.

The physiology of removing tears from the eye was more than the mere passage of tears.

DR. A. G. TALBOT (Auckland) said that the removal of the lachrymal sac was sufficient in his experience. He had done several intranasal operations, but had found difficulty afterwards in keeping the passage clear as fluid regurgitated just as easily into the sac as into the nasal cavity.

DR. H. F. SHORNEY (Adelaide) thought that the operation to be performed in these cases should be the one which gave the most constant and favourable results. His experience had been that the treatment of the sac along with the palpebral part of the lachrymal gland gave most favourable results in most cases. If the epiphora continued, he removed the orbital portion of the lachrymal gland.

DR. A. J. HALL (Dunedin) held that before operating the sac itself should be attended to. He said that in these conditions the musculature of the sac was impaired owing to the swelling and unhealthy condition of the sac. He thought that no form of treatment was successful unless the sac was treated and restored to a satisfactory state.

DR. F. ANTILL POCKLEY (Sydney) said that in his experience removal of the sac was quite satisfactory.

DR. GEORGE FENWICK (Auckland) considered that the members were unduly pessimistic in their attitude towards operations of the West type; in his experience the immediate result was the final result in the great majority of patients. Hæmorrhage was the difficulty during operation. Infiltration of the nasal wall with "Novocain" and "Adrenalin" might eliminate this. Packing he found was unreliable.

SECTION X.—NAVAL AND MILITARY MEDICINE AND SURGERY.

MEDICAL SERVICES IN THE FIELD: WITH SPECIAL REFERENCE TO THE BATTLE OF MESSINES.

BY COLONEL F. A. MAGUIRE, D.S.O., A.A.M.C.,
Deputy Director of Medical Services,
Second Military District,
Australian Military
Forces.

The medical service in the field is based on the principle of rapid evacuation of the sick and wounded. The efficiency with which this system is organized and administered, greatly affects the mobility and moral of the army.—[Field Service Regulations, Part I.]

THIS statement, which summarizes the fundamental principles which must guide all medical officers in the field, is laid down both in field service regulations and Royal Army Medical Corps training as a necessary preliminary. It is the touchstone by which we may judge whether our schemes for the handling of any military situation are sound. Rapidity of collection and evacuation of the disabled personnel is therefore the ideal which we must always keep before us.

An essential to rapid evacuation is thorough organization. There must be sound discipline throughout the medical services, together with a sense of loyalty and cooperation between the various medical units. "The game is more than the player of the game and the ship is more than the crew." If every officer, non-commissioned officer and man is moved by the knowledge that his work is helping the whole organization to run smoothly, a great deal is gained.

There are other factors, however, to be borne in mind in order to give the wounded man the best chance of recovery. It is necessary that provision should be made for warming the wounded. The wounded man is generally suffering from shock and from loss of fluids. His vitality is depressed and the reaction on his nervous system is severe. So arrangements must be made for *rechauffement* at the earliest possible moment and for the maintenance of warmth as far as possible during transport. Hot fluids are invaluable aids in the treatment of shock and can be administered freely in most cases. Gentleness in handling diminishes the bad effects of transport to a great extent.

I may summarize the main requirements for field work as follows.

1. Rapid evacuation is obtained by (i) well thought out plans for evacuation, made as far as possible beforehand or good staff work, (ii) efficient organization and sound discipline in the units in charge of evacuation, (iii) liberal transport facilities, provided extra to those of mobilization equipment. This includes the provision where possible of extra mechanical transport, preferably light railways or tramways on account of the greater comfort and speed of evacuation and an ample supply of ambulance cars, lorries and transport wagons, (iv) a minimum of delay at each stage of evacuation, which requires that wounds should be redressed as little as possible at intermediate dressing stations, (v) systematic supervision along the lines of evacuation to avoid or overcome temporary blockage with the resulting banking up of patients.

2. The provision of warmth requires (i) ample supplies of blankets right forward to regimental aid posts, (ii) organization of means to return a blanket for every blanket sent down, (iii) *rechauffement* by means of "primus" stoves or other available sources of heat at regimental aid posts (if sufficiently roomy and sheltered), advanced dressing stations and main dressing stations, (iv) hot bottles and bricks with patients on stretchers, (v) warming of ambulance cars.

3. Ample supply of hot fluids is essential at all stages of evacuation. It can best be supplied in the form of meat extracts, tea and coffee, the latter being well sweetened where possible.

4. The gentle handling of patients is essential. In the training of stretcher bearers of battalions and field ambulances, of motor drivers and of nursing duty personnel emphasis should always be laid on the necessity for gentleness and the avoidance of all unnecessary jars and jolts in the transport of wounded at all times.

On reviewing this summary it will be found that the work of the medical services in the field calls for good staff work, ample supplies and thorough training of personnel.

Staff Work.

The administrative medical staffs in armies, corps and divisions are the Director of Medical Services,

the Deputy Directors of Medical Services and the Assistant Directors of Medical Services with their deputies.

It is axiomatic that the medical staff should keep in the closest touch with the "G" "A" and "Q" branches of the staff and be, as far as may be, in the confidence of the General Officer Commanding. If this is so, they are in a position to make the necessary medical arrangements and to dispose of the medical units to the best advantage. It is important that medical operation orders should be drafted carefully, conveying all the information that it is necessary for commanding officers to know and avoiding ambiguity. Carelessly drafted operation orders will quickly produce confusion. When feasible the staff officers should look over the ground and familiarize themselves with the possible routes of evacuation. They are then in a position to deal with any variations which may arise during the operations. By liaison with the "Q" staff ample supplies of extra materials are provided and dumps of stretchers and blankets are prepared. Everything that it is possible to foresee, should be provided for, for the main work of the staff must be done before the battle. It should be completed by Z day and zero hour.

Supplies.

The medical and surgical supplies provided by the mobilization tables are ample for the actual surgical needs of the dressing stations and regimental aid posts. But the extra supplies to be drawn from ordnance, from the advanced depôt of medical stores and from auxiliary services such as the Red Cross, comforts funds *et cetera* are essential to the successful handling of many wounded. Experienced ambulance commanders and quartermasters will avail themselves liberally of these resources.

Training.

The more thoroughly trained the medical units are and the higher their moral, the better and more rapid will be the evacuation, while the wounded will be handled with gentleness and consideration. It is of importance that the bearers should be of the best physique and in good physical condition, while non-commissioned officers and nursing duty personnel should be thoroughly competent. The medical officers are then more free to attend to their professional duties with a minimum of administrative cares.

With these general principles in mind let me apply them to the particular circumstances of the Battle of Messines, which was fought early in June, 1917. In this operation Australian and New Zealand troops cooperated and the medical services of the New Zealand and Third and Fourth Australian Divisions worked together. It was thought, therefore, that it would be an appropriate operation to review, especially as many of those present actually took part in the battle and can speak from personal experience.

There is one outstanding feature about the Battle of Messines which must be emphasized at the outset. It stands out as a perfect example of a "set piece." Plans were laid for it many months ahead, so that preparations could be developed on an elaborate scale. There was a definite set objective, limited in extent, involving the seizure and holding of a piece of enemy country and a straightening out of the line. There was no intention of a general advance following the battle, so that there was no need for a general scheme for rapid advance or a pursuit battle. It must be regarded, therefore, as a particular case, that of an isolated battle with limited objective.

The battle was fought by the Second Army, under the command of General Plumer. The army was composed of three corps, the Second Anzac, the Ninth and the Tenth. Surgeon-General R. Porter was Director of Medical Services of the Second Army, with his headquarters at Hazebrouk. Colonel Mackie Begg, N.Z.M.C., was Deputy Director of Medical Services of the Second Anzac Corps.

The object of the battle was to capture the Messines-Wytchaete Ridge, to advance the line about two and a half miles in the centre of the attack and thus to straighten out a deep salient in our line as a preliminary to further attacks on the Ypres front later in the year.

The Tenth Corps was on the left, holding a front of about three miles from Zillebeke, immediately east-south-east of Ypres, to St. Eloi. The centre of the army area was held by the Ninth Corps, which extended from St. Eloi for two and a half miles to just north of Lindenhoek. The southern sector was held by the Second Anzac Corps, with a frontage of six miles, from Lindenhoek on the left to the River Lys just north of Armentières on the right.

The general arrangements for the medical services were similar throughout the army. Corps main dressing stations were established, two to each corps. The Assistant Directors of Medical Services of divisions were responsible for clearing the wounded from the line to the corps main dressing stations and the corps collecting posts. The Deputy Directors of Medical Services of corps were responsible for clearing these stations to the casualty clearing stations. From the casualty clearing stations the Director of Medical Services of the Army supervised the clearing of the line of communications.

In the Second Anzac Corps there were five advanced dressing stations, at the Brewery, Charing Cross, Underhill Farm, Lindenhoek Farm and Kandahar Farm. Owing to the physical features of the country, in which a ridge ran east and west from Hill 63 to Bailleul and divided the corps area into two at right angles to the line, Colonel Begg arranged two lines of clearance. The one south of the ridge included the Brewery, Charing Cross and Underhill Farm advanced dressing stations, which cleared the right half of the line including Ploegsteert Wood. This sector was cleared to the corps main dressing station at Pont d'Achelles where there was also a corps collecting station. The advanced dressing stations in the northern sector, Lindenhoek Farm and Kandahar Farm, cleared to

the corps main dressing station and casualty clearing post at Westhof Farm.

The corps main dressing stations were prepared some time in advance. They were well hutted, with good roads and ample room for loading and unloading cars. (There is a model of one of these stations in the Australian War Museum.) They were staffed by six tent subdivisions of field ambulances, one being drawn from each ambulance of the four divisions, thus leaving the ambulances mobile and under their own command.

There were eleven casualty clearing stations in the army area. Of these the Second Australian Casualty Clearing Station was at Trois Arbres, near Steenwerck, less than a mile from the corps main dressing station at Pont d'Achelles and the Fourth Casualty Clearing Station in Bailleul, about two miles from Westhof Farm Corps Main Dressing Station and No. 11 Casualty Clearing Station at the railway siding south of Bailleul, available to both corps main dressing stations and about two miles from each. The remaining casualty clearing stations were distributed to the other corps.

The Third Australian Division held the right sector till June 12 and was responsible for the clearance of its own area during that time. The Fourth Australian Division passed through the New Zealand and Twenty-fifth Divisions on the evening of June 7 and took over the front of the left sector on June 9. The passing through of the Fourth Australian Division called for special medical arrangements. The New Zealand Division cleared its own area and then assisted in clearing the Fourth Australian Division from the point where it took over. But the Assistant Director of Medical Services of the Twenty-fifth Division arranged for a series of relays or "leapfrogging" by which battalions as they advanced cleared back over their own area to where the preceding battalion was placed instead of clearing right through the divisional area. Owing to loss of contact during the stress of the battle this system broke down, causing a congestion of wounded and adding very much to the difficulties of the Assistant Director of Medical Services, Fourth Australian Division.

The general medical arrangements for the Second Anzac Corps are published in detail in the "Official History of the War; General History, Medical Services," Volume III, and are clear and concise. All records were kept at the corps main dressing stations and antitetanic serum was administered there. Full use was made of the liberal systems of trench tramways and Decauville railways which had been built throughout the army area. Arrangements were made at each main dressing station and collecting station for a non-commissioned officer to see that every ambulance car on unloading was equipped with its full complement of stretchers and blankets. Large dumps of these were provided at each station.

The zero hour was 3.10 a.m. on June 7, 1917, when nineteen large mines were exploded simultaneously under the enemy lines and the troops attacked behind a barrage. The evacuation of the wounded was very rapid. There had been a heavy gas bombardment of Ploegsteert Wood during the assembly of the troops and the corps main dressing station at Pont d'Achelles was kept busy before zero hour with these cases. By 6.30 a.m. 500 men had passed through this station, including 200 gassed soldiers, while 168 had passed through Kandahar Farm. The first ambulance train left Bailleul at 7.30 a.m. The wounded passed through to the casualty clearing stations rapidly, as the distances were short and the corps main dressing stations organized for rapid handling. But the distance was so short between the corps main dressing station and casualty clearing stations that there was a great tendency for overhandling of wounds. This was kept in check as much as possible. In the three days, June 7 to 9, there were 8,867 casualties in the Second Anzac Corps, 3,605 in the Ninth Corps and 5,087 in the Tenth Corps, a total of 17,559. The casualty clearing stations in Bailleul rapidly became congested on June 7 and this blocked the clearance from the left casualty main dressing station of the Second Anzac Corps. At 3 p.m. Colonel Begg diverted all the wounded of the Corps to Pont d'Achelles which received from the Kandahar and Lindenhoek advanced dressing stations as well as the southern ones. This continued for some hours and Pont d'Achelles General Main Dressing Station put through over 5,000 men, walking, sitting and lying. By 6 a.m. on June 9 Kandahar Farm had put through 1,121 walking, 799 sitting and 1,029 lying patients. The total thus of the advanced dressing station's left sector to June 9 was walking 980, sitting 1,620 and lying 1,230, total 3,830, including prisoners of war. After June 9, attacks and counterattacks ceased and the position was consolidated and held as in ordinary trench warfare.

It is interesting to note the proportions of various types of wounded in this battle as given in the official records of divisions.

	Walking.	Sitting.	Lying.
New Zealand Division, June 7 to 9 ..	26	32	42
Twenty-fifth Division, June 7 to 9 ..	61	38	
Nineteenth Division, June 7 to 9 ..	69·3	30·7	
Forty-first and Twenty-third Division	69·8	30·2	

This does not, however, show in all cases how many sitting wounded there were.

There were some new factors introduced into the medical services at the battle of Messines. Mustard gas was encountered for the first time and it had to be studied and met by new means of defence. But most of the gas casualties were from phosgene. Oxygen apparatus was supplied to each corps main dressing station and special provision was made for treating gas casualties. Shell shock cases were not evacuated as such, but were sent to a special casualty clearing station for investigation.

The general principles which were outlined earlier in the paper, may now be examined and applied to this battle.

The staff work throughout was excellent. There was close liaison between the medical staff and the general and "A" and "Q" branches of the staff.

As a result the medical arrangements were made well ahead on a well thought out plan. There are, however, two points open to criticism. The casualty clearing stations were too close to the corps main dressing stations or conversely the corps main dressing stations were arranged on such an elaborate scale that they were practically advanced casualty clearing stations. This, of course, was due to the fact that the battle was a "set piece," with a limited objective and with ample time available for elaborate preparation. Under such circumstances it was only right that the best arrangements possible should be made, but it should not be taken as a model for conditions in more mobile warfare, where it is necessary to have divisional arrangements with divisional main dressing stations that can be changed quickly, closed and moved to another situation with a minimum loss of time.

The arrangement for "leapfrogging" in the Twenty-fifth Division was unsatisfactory. It is better for each division to be entirely responsible within its own area when units are moving forward through one another. The arrangements for liaison must be most carefully thought out and supervised by a responsible officer.

As a result of the careful staff work the evacuation was very rapid from zero hour on. The only delay occurred at the casualty clearing stations in Bailleul and was largely due to the poor approaches and the congestion of traffic. This reacted on the left corps main dressing station and caused the deflection of all the evacuation to the right corps main dressing station, which fortunately was large enough to meet the situation. Otherwise a serious blockage would have arisen. It emphasizes the necessity for the provision of ample facilities for traffic.

The discipline of the units was excellent. The training and experience they had had made for the highest efficiency.

There were ample transport facilities. A main ambulance corps was available for each corps with in addition as many lorries as were required for rapidly clearing the walking wounded.

The wounded were handled as little as possible. When it was considered safe they were marked "O.K." at the advanced dressing station and were then passed through the corps main dressing station without redressing, receiving only antitetanus serum and being recorded. There was, however, a great temptation for the surgeons at the corps main dressing stations to do too much, on account of the facilities provided there. The proximity of the casualty clearing stations made this unnecessary.

Rechauffement was used where necessary. During the day the weather was fine and hot, but the nights were cold. Ambulance cars were warmed and there were plenty of blankets. The Y.M.C.A. and comfort funds of New Zealand and Australian divisions rendered most valuable assistance. Provision was made for return of blankets with cars.

GENERAL SIR DONALD J. McGAVIN (New Zealand) expressed his interest in the paper submitted as at the operations under review he was Assistant Director of Medical Services of the New Zealand Division. He agreed that corps main dressing stations had been unnecessary at Messines, as advanced dressing stations could have done all that was necessary. The position of the former had been vulnerable and extra handling had been necessary. Their institution would have been advisable (i.) if the carrying to the casualty clearing stations had been too long or (ii.) if long distance shelling had been too heavy. The only delay experienced had been at Westhoek, where cars had waited as long to be unloaded as it would have taken to have gone to Bailleul and back.

He considered that the advantage of previous knowledge and of set objective had made for the excellent arrangements which eventuated. He dealt with the question of the order that ambulance cars should not go to the front line. He estimated that the exposure of wounded to shell fire was much less with ambulances than with hand carrying. The proportion was 1 : 32. He submitted that it was more important to keep roads in good order than to maintain light railways which were easily put out of commission.

In the operations under review there was not so much shelling of the Neuve Église-Wuhinghen Road as had been anticipated.

Bearers, drivers and others had been supplied with body shields which however had often been discarded. Undoubtedly these shields had saved many lives. One peculiar fact noticed regarding them was that the khaki covering often prevented wounds after the steel plate had been penetrated.

LIEUTENANT-COLONEL J. H. HARDIE NEIL (Auckland) emphasized the perfection of the medical organization at Messines. In his opinion the only thing lacking had been a lack of facilities for treating abdominal wounds and mentioned that he had suggested their institution after the first battle of the Somme. This suggestion had been adopted later. The only thing against their establishment was their liability to shell fire, but he did not think that this was of much moment, because the location of such stations was usually carefully selected.

He referred to the improved treatment of fractured thighs with Thomas's splints and the reduction of the mortality to 15%.

With reference to stretcher bearers whom he considered should be regarded as specialists, he mentioned that adequate rest for these men was greatly to be desired.

COLONEL J. S. PURDY (Sydney) stated that it was interesting to note that as a result of experience at Messines and Passchendale, when the Canadians took over the offensive at the latter place, Colonel Ross, D.D.M.S., Canadian Army Medical Corps, eliminated the corps main dressing station and evacuated direct from the advanced dressing station at Ypres to the casualty clearing station at Remy Siding.

An excellent arrangement at Messines was the pooling of the stretcher bearers of the three field ambulances, one having an officer commanding of the forward area, another officer commanding of transport and the other at the main dressing station. To lessen fatigue and prevent stretcher bearers from being continuously at work, they were divided so that from zero for twelve hours half were at rest; thus men were not continually at work for six days without reasonable spells. At Charing Cross Advanced Dressing Station a number of gassed soldiers caused difficulty. These casualties were due to insufficient training in the use of gas masks. This was later remedied by the institution of gas schools for the proper instruction of troops.

One feature of the battle of Messines which was not, however, specially the concern of the medical service, was the delay in the burial of the dead. This was due to the late arrival of the officer concerned.

LIEUTENANT-COLONEL A. R. D. CARBERRY (Wellington) spoke of the advantages to be gained by the establishment of corps main dressing stations as a relay between the advanced dressing stations and the casualty clearing stations. These advantages he considered were as follows:

(i.) The regulation of the flow of wounded to the casualty clearing stations where the work done was limited by the capacity of the operating teams. Nearly one-third of the wounded required operation and the sorting of the various types of casualties should be carried out at main dressing stations.

(ii.) The requirements of urgent surgery, such as the arrest of hæmorrhage and emergency amputations, were best attended to at main dressing stations, while restorative measures for shock, such as blood transfusion, the application of warmth and rest, could be applied in the same place.

(iii.) The administration of antitetanic serum in his opinion was best carried out at main dressing stations; the necessary clerical recording work, the preparation of "A.F. 36" and the notification to the assistant directors of medical services of the nominal roll and disposal of the wounded of the respective divisions were also essential functions of the corps main dressing stations.

MAJOR K. GORDON (Te Aroha) mentioned that he had been a regimental medical officer at Messines and put in a plea that regimental stretcher bearers should also be regarded as specialists and be accorded special treatment.

MAJOR F. L. BIGNELL, D.S.O. (Lismore) stated that from the point of view of the regimental medical officer supplies, especially dressings, were not adequate and advocated the sterilization of emergency dressings by regimental medical officers.

COLONEL R. B. SMYTHE, D.S.O., Adjutant-General, New Zealand Staff Corps, spoke as a combatant officer and pointed out that, although Messines had a set and limited objective, medical organization in the future must be directed generally to providing for operations without such set objective. He commented on General McGavin's remarks regarding the time of evacuation of wounded and their exposure to shell fire. He stated that he thought that when an area was being covered by enemy fire, but individual targets were not aimed at, it was certainly advantageous to get the wounded away in cars as suggested, but that where fire was being directed at individual targets, it would probably be better to employ stretcher bearers, because the ambulance car made a much more conspicuous target.

MAJOR GENERAL G. W. BARBER, D.G.M.S., Commonwealth Military Forces, who presented Colonel Maguire's paper, stated that the experience gained at Messines was invaluable to him as later he had been responsible for the arrangements at the battle of Amiens and of the Hindenburg Line and had based his orders on the knowledge gained. In traversing the comments of previous speakers he mentioned: (i.) That light ambulance cars later ran as far as regimental aid posts. His own opinion was that light railways were extremely vulnerable and alternative methods had to be provided, (ii.) forward operating units were later instituted by creating resuscitation teams for each division. He also dealt with the criticisms of main dressing stations and agreed that the abolition was justified in certain circumstances.

FRIDAY AFTERNOON, FEBRUARY 4, 1927.

COMBINED MEETING.—SECTIONS I AND IV.

TRANSITION OF SIMPLE TO TOXIC GOITRE.

By S. V. SEWELL, M.B., Ch.B. (Melbourne),
Honorary Physician, Melbourne Hospital.

IN examining the question as to the cause or causes which lead to thyreo-toxic states in goîtrous patients, it seems pertinent first to investigate the conditions which lead to so-called physiological enlargement in non-goîtrous subjects. It is important in this connexion to realize the extreme vascularity of the normal thyreoid. It has been carefully estimated that four times the amount of blood passes through the thyreoid as compared with the kidney in twenty-four hours and since the amount passing through the kidneys is from 700 to 1,000 litres, this means at least 2,800 litres for the thyreoid. It is probable that an organ with such a large blood supply has other important functions than the manufacture of the minute quantity of thyroxin which is necessary for its action as a catalysing agent, five milligrammes or one-twelfth of a grain of which given intravenously being sufficient to control a gross myxœdema for from three to four weeks. It is quite possible that one of its great functions is to act as a detoxicating mechanism as the blood passes through it.

The stresses of puberty, menstruation, pregnancy and the menopause would seem to be met if the mechanism is normal by temporarily increased vascularity and so an increase of function. Should the gland be unhealthy, owing to low-grade sclerosis resulting from the various infections of childhood, septic tonsils and the like, the increased vascularity may be insufficient and actual cell hyperplasia or overformation of colloid results and a definite goître becomes palpable.

Such cell hyperplasia may be locally placed in areas of interstitial irritation and adenomatosis is the result or definite adenomata are formed. During the late middle period of life, atrophic changes are slowly occurring in the essential thyreoid tissue and when an insistent call occurs, causing increased thyroxin consumption, the somewhat atrophic gland tissue is unable to supply the increased demand in an adequate manner. The newly formed adenomatous tissue begins to produce thyroxin actively and since it is outside nervous influences thyroxin production runs mad and hyperthyreoidism results. Such an increased call for thyroxin may be the result of bacterial infection or of toxins produced from septic foci or again the hyperactivity of the adenomatous tissue may have been caused by the injudicious use of iodine which should not be employed in the treatment of adenomatous cases in the atrophic period of life, though its use in cases of adenoma associated with colloid goître in early life is quite permissible, because the thyreoid generally is not atrophic and responds adequately with increased production of thyroxin.

Intense psychic stimulation, associated probably with a great rise in basal metabolism, may be adequate to change a simple adenoma into adenoma with hyperthyreoidism and occasionally may even cause an apparently normal gland to undergo hypertrophy and hyperplasia producing a most acute form of Graves's disease. Some years ago a serious railway accident took place in Victoria. A great number of people were killed and injured. Two patients who came under my observation will illustrate the importance of this psychic factor. One, aged twenty-six, was a confidential clerk and her employer assured me that up to the time of the accident she was efficient, quiet, self-contained and apparently had no thyreoid abnormality; yet when seen by me ten days after the accident she presented all the signs of Graves's disease in its most exaggerated form. This patient within a year made a good recovery without operative interference.

The second case was that of a woman in the middle forties with an obvious adenoma of the thyreoid which had been present for many years,

but with no symptoms suggestive of hyperthyreoidism. This patient was seen by me in consultation three weeks after the accident and presented definite signs of hyperthyreoidism. Medical treatment in this case produced little amelioration of symptoms and ultimately an operation was performed with good results.

Anyone who has seen the striking effects produced by the exhibition of adequate doses of iodine in cases of exophthalmic goître, must feel that a relative insufficiency of iodine is bound up with the symptom complex. The transformation of a soft, pulsating gland turgid with blood into a firm, hard gland almost suggesting malignancy at the end of a few weeks of iodine treatment can leave no question as to its effectiveness. Plummer's theory of dysthyreoidism with an incompletely iodized thyroxin molecule is extremely suggestive and as a working hypothesis is of the greatest value; for early cases properly managed iodine treatment and dealing with septic foci may still prove to be adequate and only the general practitioner with his opportunity of seeing patients in the early stages can answer the question. However, by the time the consultant sees patients with Graves's disease the manufacturing plant for thyroxin has been so much increased by hypertrophy and hyperplasia that common sense demands iodine treatment as well as a surgical reduction of the amount of thyreoid tissue.

Any review of this subject which neglected the very suggestive work of Williamson, would be very inadequate and should his thesis be substantiated by the work of other observers, a distinct step forward will have been made in the understanding of the complicated thyreoid problem.

According to Williamson's work the basic architectural feature of the thyreoid is a lymph sinusoid which carries the secretion of the gland to different lymphatic vessels which pass from the hilum and are joined by the lymph vessels from the parathyreoid on their way to the thymus. It is suggested that between periods of peak demand, thyroxin storage largely takes place in the thymus and not in the thyreoid itself. He suggested that the circulation of the colloid is a necessity as a preliminary to formation of normal thyroxin. It is further suggested that acute conditions of hyperplasia and hypertrophy of the gland cells may partially block the sinusoid and so direct absorption of thyroxin into the blood stream comparable to the mechanism of an obstructive jaundice results.

Such an incompletely elaborated thyroxin may be immature and toxic, not having passed through the thymus where possible changes may occur in its constitution.

He would explain the crises of acute Graves's disease as occurring in this fashion. Late or secondary Graves's disease he would ascribe to chronic cirrhotic changes in the interstitial tissue leading to partial blockage of the sinusoids. Compensatory hypertrophy and hyperplasia of gland tissue occurs as in cirrhosis of the liver. Later when some insistent call for thyroxin occurs, the lymph pathway being more or less blocked, regurgitation into the blood stream takes place with resulting thyreotoxicosis.

Should Williamson's work prove to be substantially correct, it will in no way invalidate Plummer's conception of the dysthyreoidism of Graves's disease and its possible causal association with the presence of an incompletely iodized thyroxin in the blood stream.

THE MORBID ANATOMY OF THE THYREOID GLAND IN HYPOTHYREOIDISM AND MYXŒDEMA.

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THE term hypothyreoidism includes all conditions due to defective thyreoid function. That myxœdema is the more severe form of hypothyreoidism has been borne out by metabolic rate estimations.

We propose to deal only with certain aspects of the ætiology and the pathology. Congenital absence of the thyreoid is very rare and in a group of eleven patients with the condition, mostly females, all were high grade cretins, none became sexually developed and all died early. Patients with thyreoaplasia reaching adult life probably have small nodules of thyreoid tissue at the root of the tongue; when the thyreoid is rudimentary, aberrant and accessory tissue may functionate (Janney[1]). We need only to mention hypothyreoidism following resections of the glands or following destruction by suppuration and, omitting altogether endemic cretinism, we shall pass on to the hypothyreoidism of even more obscure causation. The ætiological relation of the infectious diseases of childhood is usually difficult to demonstrate because symptoms may not develope till after many years. Nearly all the infectious diseases of adults have been said to have been followed by hypothyreoidism; chronic intoxication, such as lead poisoning and alcoholism, have been blamed and psychic states of grief, anxiety and so forth may precede the condition. There is no doubt that hereditary factors are sometimes a striking feature, as for example, in the three hypothyreoid children whose father manifested dysthyreoidism, instanced by Janney. If we turn to the textbooks of medicine to ascertain the morbid anatomy of myxœdema, we shall find little but the word atrophy, or that the gland is very small or almost absent or that the gland shows fibrosis. In Osler and McCrae's "Principles and Practice of Medicine," we find Riedel's thyreoiditis or struma dealt with in a

separate section from myxœdema and there is no hint of their relations. It is perfectly obvious, of course, that the morbid anatomy as so described is that of the latest stages of a hypothyreoidism which has been present for a varying period, often for many years, and the material must have come in great measure from the *post mortem* room. Until recent years it is doubtful whether the histopathology of the gland in the early stages has been recognized and even now it seems to us that there is much that needs correlation. But there has come new light on the histopathology, mainly from thyreoid tissue removed by surgeons. This era began in 1896, when Riedel reported his first case of iron-hard struma or chronic strumitis. Up to 1910 only seven cases had been reported and Riedel himself had seen only three patients in a large surgical goître experience (1,064 goîtres). Its general bearing upon the subject of myxœdema was apparently not suspected. In 1910 Riedel published a further communication[2] on this subject. He described a typical case, that of a young man of twenty-nine years, previously healthy, in whom a lump had developed in his neck first on one side, then on the other and in six weeks he was dyspnœic and had choking attacks. There was no fever. Operation disclosed the carotids and jugulars adherent to the swollen gland and lateral lobe excision was impracticable. A wedge-shaped piece was excised and in this there was infiltration with round and spindle cells amongst the gland elements. There was slight endarteritis as in many chronic inflammatory processes. In places there was a strong growth of fibrous tissue. The patient had a protracted illness with recurring attacks of dyspnœa for some years, but was then after fourteen years quite well. Riedel went on to discuss a patient whose illness was reported by Silatchek[3] and who recovered spontaneously. "Does this give the clue to the condition's rarity. Do cases occur more often but only reach a certain stage and then recover so that they are not seen in surgical clinics? Perhaps practising physicians see these cases occasionally, but only when the whole gland is affected or the inflammation is severe, is surgical aid sought." In the condition he describes, only a wedge-shaped piece can be resected. The gland bleeds freely, but it is not serious as the vessels have only a small lumen. The ætiology of this disease is a mystery. As to diagnosis, if the condition be not borne in mind, many patients might be sent away with a diagnosis of inoperable carcinoma.

Since Riedel's reports French writers have reported similar cases as ligneous thyreoiditis and Hashimoto[4] in 1912 described a condition of intense lymphoid infiltration in the thyreoid as *struma lymphomatosa*. Ewing[5] saw further patients and concluded apparently correctly that this was the early stage of the condition described by Riedel. Ewing describes the lesion as benign granuloma of the thyreoid, a term which is perhaps too suggestive of a purely hypothetical ætiology. Quite recently several papers have appeared on this subject. Shaw and Smith[6] review twenty-three reported cases of Riedel's chronic thyreoiditis and add six cases seen by them. Smith and Clute[7] add reports of five further cases under the title chronic ligneous thyreoiditis. Williamson and Pearse[8] give us the impression of having seen a number of cases and discuss the condition under the title of lymphadenoid goître. Meeker[9] describes one case and in reviewing the literature cites cases not included by Shaw and Smith, so that it would seem that up to 1926 about forty-seven cases have been reported and we have added four cases of patients seen in Sydney. The condition is not common before the age of thirty, though two patients were twenty-three; the age of the majority of the patients lay between thirty and sixty. About 75% of the patients were women and there appears to be no relation to the menopause; ætiological factors are not at all obvious. Syphilis and tuberculosis seem to be definitely excluded. The common symptoms are enlargement and dyspnœa. The most common preoperative diagnosis was malignant disease of the thyreoid. At operation the gland was found in many cases adherent to the trachea, the muscles and even to the carotid sheath. This extracapsular extension leading to a matting together of the tissues is, however, not always recorded and often a lobe or the whole gland has been successfully resected. The duration of the enlargement before consultation may be only a few weeks or may be several years and it may be generalized or of one lobe or even apparently focal. The condition is in itself benign, but deaths have occurred from complications following operation. In all these reports the histological examination when made revealed various grades of small round cell infiltration and sclerosis and there appears to be little doubt of the close resemblance of the histological findings in reports from different writers. It is certainly uncommon to find any reference to hypothyreoidism or myxœdema before operation. Basal metabolic rates have been worked out apparently in very few patients either before or after operation. Smith and Clute reporting on their five patients state that of four submitted to partial thyreoidectomy, three subsequently developed myxœdema, judged both clinically and by basal metabolic tests; they held that patients must unquestionably be followed up afterwards with this possibility in mind. It is their belief that partial thyreoidectomy for Riedel's struma will be followed by myxœdema in a very large proportion of patients. It will be shown that the basal metabolic rates were worked out in three of our four patients and the results were – 8%, – 21% and – 15% respectively. In the first there was clinically no evidence of myxœdema. In the second some exophthalmos was present without von Graefe's sign, she had a drawling and thick voice, the skin of her hands was scaly but warm; there was falling of hair, but no sensation of cold.

The third patient felt both heat and cold, there was thinning of the hair of the eyebrows, a hoarse voice, parchment-like skin of the forehead, a sallow complexion, dryness of the skin of the hands and arms.

Leaving this very interesting surgical contribution to the subject of hypothyreoidism, we shall divert your attention to a very important paper by Simmonds[10] which has apparently been overlooked by recent writers on this subject. Simmonds turns again to *post mortem* material and reports upon twenty thyreoids found to be affected by varying grades of what he calls "chronic thyreoiditis and fibrous atrophy." This paper follows on another published in 1916[10] in which he dealt with the affections of the thyreoid in acute infective diseases and in which he remarked upon the rarity of chronic inflammations with functional defects as sequelæ of the acute conditions. The subjects were all women, the youngest forty-four; only four of the twenty were under fifty, the average age was fifty-seven. The twenty cases are subdivided into four groups of five showing diminishing degrees of the lesion. The first and most advanced group of five patients were all old women and two had myxœdema and two others manifested loss of pubic and axillary hair. One patient had syphilis. The thyreoid gland at autopsy was found to be of small size and compact, the cut surface greyish-white, with no normal looking areas; in one case the gland was almost unrecognizable. This naked eye picture resembles that of the textbook gland in myxœdema, but when we come to the microscopical report we find the following. There was no normal gland tissue, much fibrous tissue containing small blood vessels, in the media of which hyaline changes sometimes going on to calcification were noted. The connective tissue is sometimes acellular, sometimes beset with groups and "streaks" of lymphocytes, plasma cells and fibroblasts and exceptionally there are germ centres. In many places within and without round cell infiltrations, there are peculiar rounded, oblong and irregular groups of epithelial cells or "cell complexes" (syncytial masses), cells with large nuclei and well stained cytoplasm and there is no demarcation between adjoining cells. Sometimes they form columns containing very small drops of colloid. Like those writing on Riedel's thyreoiditis, he interprets the epithelial cell masses as an attempt at reparation and restoration of the destroyed alveoli, in other words, a form of compensatory hyperplasia. He supports the suggestion of Lubarsch that perhaps the lymphocytic infiltration is related to repeated inflammatory events. The atrophy is a result of chronic inflammation. The lymphocytes cause a dissolution of the alveoli, only fragments remain and these attempt compensatory overgrowth. The lymphocytes are the cause of the atrophy, not the result. The condition described might be called cirrhosis of the thyreoid without prejudice to its ætiology (see Figure VII, 1 and 2).

Of Simmond's second group of five patients, none had myxœdema, two had syphilis, and the thyreoid gland at autopsy showed a lesser degree of the same lesion, namely infiltrations with lymphocytes and plasma cells, fibrous tissue overgrowth and disappearance of parenchyma. In the third group none had myxœdema, one had syphilis and there was only partial destruction of epithelium and other tissue of the thyreoid gland. In the final group of five patients, none had myxœdema and none syphilis; to cite one case, for example, there was slight diminution of colloid in the thyreoid gland, numerous small, round cells and germ centres amongst the alveoli and slight increase of fibrous tissue.

In all these twenty cases the differences are quantitative and not qualitative. In the first five only had the process gone on practically to complete destruction of the parenchyma and yet only two of the five were recognized as myxœdematous and none of the fifteen less advanced cases. General adiposity was repeatedly met with in these patients and Simmonds remarks that this adiposity should be regarded as of purely thyreoid origin. Simmonds concludes by saying that chronic inflammatory events occur in the thyreoids of old women not seldom. They are revealed by small, round cell infiltrations, sclerosis and so forth. This process goes on only in a small number of cases to severe thyreoid deficiency as revealed by myxœdema, but is more often associated with adiposity. The ætiology of chronic thyreoiditis is unknown, but he thinks that infective diseases may play a rôle.

Curiously enough, Simmonds who had not seen a case of Riedel's struma, considered that this latter disease was *sui generis* and distinct from chronic thyreoiditis. But we suggest that Simmonds has been led astray by the severity of the lesions which Riedel happened to see. They were, it is true, iron-hard enlargements with periadenitis, due to the extracapsular extensions of the infiltrations, but in the later case reports reference is found to milder lesions and the striking and most important fact is that histologically the gland, whether the enlarged gland of Riedel's struma or the small glands of chronic thyreoiditis described by Simmonds, all reveal varying grades of a similar lesion which is essentially an inflammatory lesion leading to atrophy. That hypothyreoidism or myxœdema only occasionally follows is remarkable considering the degree of atrophy in some cases. It may be that as the lesion is most common in middle life and later, the small amount of compensatory hyperplasia found is sufficient to prevent obvious hypothyreoidism. But that the glandular activity is sometimes nearly insufficient can be inferred from the fact that removal of one lobe has precipitated myxœdema or as in our cases has resulted in the depression of the basal metabolic rate. The histological lesion is a specific and characteristic one comparable with but not closely resembling the lesions in the lymphatic glands and other organs in Hodgkin's disease. We suggest that various factors may determine whether the gland will present the picture of Riedel's struma or the less severe lesions of later writers or remain unrecognized as in Simmond's cases. These factors include the condition of the gland at the onset of the condition, the possible occurrence of hyperplasia as an initial lesion and the actual intensity of the small, round cell infiltration.

Granted the above conception of the essential oneness of these lesions and the occasional occurrence of myxœdema as a sequela, has the thyreoid

gland in the majority of cases of myxœdema been subject to these characteristic histological changes? In other words, we come back to the original question, what is the histopathology of the gland in hypothyreoidism and myxœdema. If it is the lesion above described, then it is obvious that this atrophy is far more common than are the advanced degrees of thyreoid deficiency, a statement which is reminiscent of that concerning hypertrophy, that it is far more common histologically than is hyperthyreoidism clinically recognized. We consider, however, that a final answer to our question cannot yet be given. What is wanted is a large series of histological reports on the gland in hypothyreoidism and myxœdema. Other types of non-goitrous atrophy doubtless occur and we have seen *post mortem* definite intralobular fibrosis of the thyreoid in a cachectic subject dying of fibroid pulmonary phthisis and tuberculous pyelonephritis. We have seen also lesser degrees of fibrous increase in other autopsy material.

The characteristic histological picture in chronic thyreoiditis is not necessarily the prerogative of this gland, for it appears that von Mikulicz's disease of the lachrymal and salivary glands presents a similar lesion; Shaw and Smith found a somewhat similar lesion in the adrenals in Addison's disease and we have seen this type of lesion in an atrophic calculous kidney. Such evidence as there is and the majority of contributors submit to this hypothesis, is in favour of the lesion being a chronic inflammatory one of unknown causation. We should mention, however, that Williamson and Pearse in their very thoughtful and suggestive monograph put forward the somewhat difficult conception of a physiological subnormal gland, undergoing compensatory hyperplasia.

In conclusion the clinician may find it difficult to correlate hypothyreoidism and myxœdema with Riedel's thyreoiditis or struma, because of the common absence of a history of enlargement in myxœdema, but the clinician would perhaps offer no opposition to accepting Simmonds' thyreoiditis as the underlying condition in the gland. But if told that advanced hypothyreoidism or myxœdema was not much commoner in one than the other and in both it was uncommon (lesser degrees of hypothyreoidism may be common, though this has not yet been demonstrated), then perhaps he would be disposed to accept the hypothesis provisionally of a specific histopathology, a lesion of the thyreoid far commoner than is the myxœdematous syndrome. Similarly the renal lesions of chronic nephritis are commoner than uræmic manifestation. Lesser degrees of hypothyreoidism associated with this specific lesion are probably far commoner and the estimations of the basal metabolic rate may prove to be of great value in this field.

Lymphadenoid Goitre Probably the Early Lesion.

Three patients were subjected to partial thyreoidectomy for enlargement of the thyreoid of a firm non-cystic character, not associated with toxic symptoms, but in two cases, possibly three, with pressure symptoms; also one typical Riedel's struma.

Case I.—V.L., female, married, *ætatis* thirty-two years at the time of the operation, had a general enlargement of the thyreoid, more on the right side than the left. There was slight interference with respiration, but no pain nor toxic symptoms. At operation the right lobe and isthmus were removed. A solid piece of gland 5 × 2 × 1·7 centimetres in size resembling the gland in exophthalmic goitre but paler was noted. It contained no visible colloid and no cysts: it was of uniform structure. There was no reaction to the Wassermann test. Two months after the operation she was reexamined. There was no recurrence. The patient then felt and looked well. The basal metabolic rate was – 8%.

The histological characters are seen in Figures I and II, low and high power magnification. A remarkable infiltration by lymphocytes replacing much of the gland tissue and a fibrous tissue increase are evident. The lymphocytes are both diffusely arranged and also form germ centres. There are plasma cells amongst the lymphocytes in the diffuse infiltration. The trabecular arrangement and gland units are distinguishable, but some units are completely replaced by lymphocytes. The glandular tissue consists of a few large acini, many small acini and solid groups of thyreoid cells, sometimes forming syncytial groups. Many acini contain deeply staining colloid, but the total amount is very small. The solid groups and small acini have epithelium which suggests activity. The capsule is thickened, vascular and contains collections of lymphocytes and the trabeculæ are thickened and contain lymph sinuses. Fibrous tissue is replacing some of the gland units. We regard the condition as an early, active stage of lymphadenoid goitre.

In Figure II, another section of the same specimen, a germ centre is seen at the top, with diffuse infiltration below; in the right lower corner a colloid acinus and just above it two very small acini.

Intermediate Stage.

Case II.—E.E., female, married, *ætatis* fifty-five years at the time of the operation, had had a generalized, very firm enlargement of her thyreoid gland for eight months. She had had a choking sensation associated with pain at first; her voice became slow; she did not feel the cold; her breathing was difficult; her hair was dry and fell out; exophthalmos was present; von Graefe's sign was not elicited. Apparently she had had symptoms of exophthalmic goitre thirty years previously after child birth. Her face had a high colour. Her voice was drawling and thick, her eyebrows were masculine, the skin of her hands was scaly but warm. The basal metabolic rate after operation was – 21%. Her serum did not react to the Wassermann test.

At the operation the right lobe was removed. It was firm and hard, somewhat lobulated on the surface and the cut surface was greyish with a distinct yellowish tinge; it was not unlike the beef-steak gland of exophthalmic goitre, except for this pale yellow colour. No visible colloid or cysts were present. Microscopically there are seen numerous greatly thickened fibrous trabeculæ and also some finer fibrous branches running into the lobules in all directions, constituting a fine as well as a coarse sclerosis (see Figure III). The analogy with hepatic cirrhosis is very striking. There is, moreover, a most diffuse infiltration with lymphocytes and plasma cells and in places germ centres, but fewer than in the specimen of Figures I and II. There are no normal colloid-containing acini. There are a few minute atypical acini containing a droplet of colloid and some in a catarrhal condition, but the thyreoid cells are mostly in irregular syncytial masses and a droplet of colloid often lies eccentrically in the mass. The cells are cubical and the cytoplasm finely granular or vacuolated and the nuclei not vesicular in appearance. We do not see any true giant cells. The resemblance to carcinoma or even epithelioma is worth mentioning. There are no fibrillæ between adjacent cells. Fibroblasts and fibrous tissue formation are easily made out. There is an interesting vascular lesion. The larger arterioles are on the whole unaffected, but in places in the smaller, intimal proliferation with narrowing of the lumen or hyaline degeneration or intimal fibrosis extending into the media,

not unlike arterio-capillary fibrosis (hyperplastic sclerosis) of the kidney is seen.

Figure IV. is a higher power view of the infiltration, showing small thick bands of fibrous tissue intimately mingled with lymphocytes, plasma cells and the remains of thyreoid parenchyma (acini) in the form of irregular syncytial masses or single, rather large thyreoid cells.

CASE III.—X., a female, in late middle life, had a portion of her thyreoid gland removed for goitre (enlargement). No special features were noted at the time. Microscopically it resembles in many ways those of Case II and the stage of the process is not greatly different (see Figures V and VI). The trabeculæ are not so thick, but the diffuse intralobular fibrosis is more complete in some areas. There is a very diffuse and generalized infiltration by lymphocytes and plasma cells and only occasional germ centres. The gland tissue is enormously reduced. There are in some areas very small acini with colloid lying amongst the infiltration, but more numerous small syncytial masses with two, three or four nuclei and an eccentric droplet of colloid. Some of the acini are catarrhal and contain small round cells in their lumina. In some situations colloid appears to be intracellular. The thyreoid cells have rather large nuclei which are not very vesicular and the cytoplasm is finely granular. There is again an interesting arterio-capillary fibrosis. Many larger arterioles are affected by concentric or eccentric intimal and medial fibrosis with or without narrowed lumen. I am at a loss to say whether it is a primary lesion or an involutionary change accompanying atrophy of the gland.

Riedel's Struma, Lymphadenoid Goitre.

CASE IV.—Mrs. F.S., *ætatis* forty-one years, with three children, when first seen had had choking attacks and hoarseness of voice for three months. She felt the heat and cold. The attacks awakened her at night and were so severe that she would jump out of bed as if suffocating. She had difficulty in swallowing hard things. She was a healthy looking woman with a hoarse voice. There was a hard mass in the position of the thyreoid, not tender; the right side was larger than left and extended down behind the manubrium and appeared to be fixed to the deeper structures. There was no fluctuation nor pulsation. No reaction was obtained to the Wassermann test. She had scanty eyebrows. The skin of her forehead was parchment-like, as were her hands and arms. The diagnosis was carcinoma of the thyreoid or malignant disease of the lymphatic glands secondary to a primary growth in the pharynx, larynx or œsophagus. An ear, nose and throat surgeon could find no primary growth, but the left vocal cord was almost fixed in the middle line.

At an exploratory operation the superficial veins were seen to have been much distended. The mass was found to be thyreoid gland, very hard and firmly attached to the trachea; the surrounding tissues were matted together. The growth was deemed to be inoperable. The wound healed up and patient was discharged. She was afterwards treated by X rays. She did not lose weight and when seen fifteen months later was heavier than before; the enlargement of the gland was greatly diminished. She had no trouble with respiration except on exertion. No lymphatic glands were palpated. Her voice was hoarse. The basal metabolic rate was – 18%.

A second exploratory operation was performed. The incision was made over the isthmus. The deep cervical fascia was greatly thickened and in the position of the isthmus was only exceedingly tough tissue which had no resemblance to thyreoid. Microscopical examination revealed very dense fibrous tissue in which were a very few small collections of small round cells. No thyreoid cells or acini could be made out.

There might be those who would say this is a case of carcinoma of the thyreoid cured or relieved by radiation, but taking into consideration the symptomatology of Riedel's struma, its usually benign course and the striking history of this patient, the absence of cachexia, the absence of dissemination, though the tumour was at its maximum for some considerable time, I venture to describe this case a typical one of Riedel's struma.

Fibrous Increase.

C.B., male, *ætatis* two months; autopsy specimens. Thyreoid weighed 1·5 grammes only. The thymus was large. This infant died with *ophthalmia neonatorum* and symptoms of gastro-enteritis. *Post mortem* broncho-pneumonia and hepatic cirrhosis were also discovered. There was great fibrous thickening of the capsule of the thyreoid and of the trabeculæ and some groups of alveoli were cut off and undergoing atrophy. Alveoli were mainly small and medium; the colloid was not very abundant. The epithelial cells were cubical and low cubical. Numerous interalveolar cells were present. Lymphocytes were not abundant (see Figure VIII).

It is difficult to explain the fibrous increase in the liver and thyreoid on the evidence available. It is possible that the infant was suffering from congenital syphilis as well as ophthalmia.

Intra-lobular Fibrosis.

G.F., a male, *ætatis* forty-two years; autopsy specimens. Death was due to chronic fibroid phthisis and tuberculous pyelonephritis. The body was wasted. The thyreoid gland was small, weighing only twelve grammes and was fibrous and adherent to the trachea. There was polylobular cirrhosis of the liver. In the thyreoid extensive fibrosis is seen, so that the alveoli are in many places surrounded by fibrous tissue (see Figure IX). The alveoli have undergone various changes; some are lined by columnar epithelium and contain pale colloid, others have no demonstrable epithelium and may contain deeply stained colloid; there are also small groups of pale cells forming minute alveoli. Some alveoli contain slate coloured, globular, altered red blood cells, others irregular lumps of colloid. In others there is desquamation. The nuclei of the cells are small and deeply stained, so that it is difficult to identify the lymphocytes.

There is undoubtedly a chronic thyreoid lesion, a cirrhosis with atrophy of many alveoli and a hyperplasia of the cells of others. It is reasonable to suggest that it is due to the chronic intoxication of tuberculosis and if so, it is an example of atrophy due to a definite intoxication and distinct from the special form known as lymphadenoid goitre.

It is not known whether there was any hypothyreoidism.

For comparison with lymphadenoid goitre I attach a reproduction of a section of a kidney with atrophy associated with calculi in calyces and pelvis, pyelitis and a leucoplakic condition of the mucous membrane of the renal pelvis (Figure X). The kidney was small and much fat was adherent to its capsule. The capsule stripped off with great difficulty leaving a pale, but not granular surface. There were three smooth, dark calculi lying in the pelvis and calyces and the atrophy of the kidney substance was considerable, the remaining cortex being mainly in the middle interpyramidal zone, the atrophy greatest in the lower pole. The mucous membrane of the renal pelvis was leucoplakic. Microscopical examination reveals some groups of tubules not greatly affected, but for the most part there is a considerable atrophy of the parenchyma associated with an intense lymphocytic infiltration with the formation of germ centres. There are numerous plasma cells in places and much smaller numbers of eosinophile and neutrophile polymorphonuclear cells. The tubules in these areas may be slightly dilated, with flat epithelium, and may contain an eosinophile substance staining like colloid or may be very small with scarcely any lumen, but with healthy looking cells or epithelial cells may be aggregated into small groups without a line of demarcation between the cells. There is also formation of fibrous tissue. The small arterioles show various degrees of intimal proliferation and fibrosis leading to occlusion of lumina.

Altogether save only for the presence of small numbers of eosinophile and neutrophile polymorphonuclear cells, it is a remarkable picture of a pathological process which is associated with atrophy of the kidney and in many respects analogous with lymphadenoid goitre.

Figure XI is from a section of an autopsy thyreoid showing no abnormalities and is for comparison with the other figures.

Summary and Conclusion.

1. Three cases of lymphadenoid goître (*struma lymphomatosa*) and one of fully developed Riedel's struma have been described. There has been a definite tendency towards hypothyreoidism.

2. The striking similarity of the microscopical picture of the above goîtrous lesions and of the atrophic lesions of "chronic thyreoiditis and fibrous atrophy" described by Simmonds, suggests their essential identity, and it is difficult to believe that the ætiology, though quite obscure, is dissimilar.

3. The history of patients presenting hypothyreoidism and myxœdema does not, as a rule, indicate the cause of the thyreoid lesion and it is suggested that a quiet pathological process with the microscopical features above described is the most common condition of the thyreoid gland in these patients.

Acknowledgments.

For access to clinical and pathological material we desire to express our indebtedness to Sir Alexander MacCormick and Dr. John Morton, Honorary Consulting Surgeons, and Drs. Abbott, Craig, Dansey, McKelvey, Poate and Storey, Honorary Surgeons to the Royal Prince Alfred Hospital, and also to Dr. Keith Inglis and Dr. Marjorie Little. Mr. Burfield and Mr. Dawes have rendered valuable technical assistance.

GOÎTRE IN GIPPSLAND.

By Walter Summons, O.B.E., V.D., M.D., D.P.H.,
Honorary Physician, The Alfred Hospital, Melbourne; Member of the State Health Commission of Victoria.

The originating idea of this paper was that it should be a survey of goître in that part of Victoria spoken of as Gippsland where endemic goître was known to exist. Consequent on that endeavours were made to collect coexisting facts, as for example the geology of the district and the iodine content of the water, with the view of preventing or treating the condition. This was being done independently of this Congress. Unfortunately, four "casualties" occurred amongst those assisting and the work has been hindered for the time being. This can thus be regarded only as a brief preliminary report.

Gippsland is the name given to the wide littoral of the eastern portion of Victoria. The greater width of the level country is ascribed to an uplift of the coast. This also has led to the formation of the Gippsland lakes. The rivers arise in the mountains and are snow rivers to begin with, but receive tributaries that flow through every variety of geological formation. A geological map was shown at Congress.

The incidence of goître is obtained from the reports of Dr. Jean Greig, the chief school medical officer, and of Dr. J. J. Harris, the district health officer, whose figures were supported by the writer's own examination of the children. Dr. Harris was to have collaborated in this paper, but ill health has unfortunately prevented his doing so.

The details of the examinations at Bairnsdale are shown in Table I.

When the goîtres are grouped according to the classification used by the Swiss Goître Commission, namely: Class 0, thyreoid gland not felt; Class I., thyreoid hardly felt on palpation; Class II., thyreoid easily felt; Class III., thyreoid easily felt and neck obviously enlarged by it; Class IV., gross deformity of the neck, the analysis of the figures gives the results shown in Table II.

At the Sale State School the figures of an examination by the writer are given in Table III.

In 1924 of fourteen boys between the ages of twelve and fourteen attending the junior technical school at Sale, eight or 57% had goître.

Bairnsdale and Sale are the two chief towns of Gippsland. Bairnsdale is on and Sale near the lakes which communicate with the open ocean. Lucknow is a suburb of Bairnsdale. The water supply of Bairnsdale is by a reticulation service drawn from the Mitchell River; the suburb of Lucknow has no reticulation supply, but tank and well water is used. Some of the goîtrous children live in the suburb and attend the high school. Certain affected children at all the schools live on the farms in the vicinity of Bairnsdale, so that they drink tank or well or dam water at home and the river reticulated water at school. The water supply of Sale is from the Thompson River almost entirely. These two towns are in the dry part of Gippsland. Moe and Traralgon are examples of the towns well away from the sea in heavily timbered country where there is

Table I.

School.	Boys.			Girls.		
	Number.	Goîtres.	Percentage.	Number.	Goîtres.	Percentage.
1. Bairnsdale State School ..	202	68	33·6	184	66	35·8
2. Bairnsdale High School ..	47	13	27·6	76	36	47·3
3. Lucknow State School ..	56	11	19·8	53	17	32·0

TABLE II.

School.	Boys.					Girls.				
	Class.				Total.	Class.				Total.
	I.	II.	III.	IV.		I.	II.	III.	IV.	
1	53	6	9	—	68	42	9	10	5	66
2	12	—	1	—	13	22	1	12	1	36
3	5	3	3	—	11	12	4	1	—	17

a high rainfall. The results of the examination of the State school children in these two towns are given in Table IV.

Goître also is prevalent in the other following centres of Gippsland: Leongatha, Warragul, Walhalla, Maffra, Stratford, Morwell, Mirboo, Dargo, Buchan, Bruthen, Tongio and Omeo. All these towns are underlined on the map.

As a contrast the school medical officers found among two thousand girls between the ages of nine and fourteen in the schools of Bendigo, Ararat, Ballarat, Warrnambool and Melbourne (Clifton Hill, Collingwood, Abbotsford) the presence of enlarged thyreoid glands in eleven or 0·5% only. These localities are scattered throughout the remainder of Victoria.

Victoria, of course, is not an old established colony and Gippsland is still a sparsely settled part. Many families have settled in the district only recently, few have lived there for more than a generation and fewer still for two generations. It has been recognized that goître has been endemic in Gippsland since the occupation by white people, but till recently no attempts have been made to determine its incidence. Inquiry from the children

TABLE III.

Age of Child.	Boys. Class.			Girls. Class.		
	0	I.	II.	0	I.	II.
5 to 8	42	38	6	24	10	—
8 to 14	59	55	7	37	51	16

TABLE IV.

Towns.	Sex.	Number of Children.	Goitrous.	Class I.	Class II.	Class III.
Traralgon ..	Boys	195	29	11	16	2
	Girls	173	37	15	15	7
Moe	Boys	71	6	2	3	1
	Girls	69	2	0	2	0

affected repeatedly elicited that elder sisters or the mother or an aunt had goître.

The Director of Agriculture in answer to an inquiry replied: "Goître is not prevalent to any appreciable extent in the animals. There are very few recorded cases in horses, cattle and sheep. None have come under the notice of the Veterinary Staff of the Live Stock Division."

The thyreoid enlargement conforms to the simple parenchymatous type. None of the children exhibit evidence of hyperthyreoidism. The physical development was up to standard and no defects were found that separated them in any way from children of other localities. The teachers are, however, of the opinion that the scholars in the Sale and Bairnsdale schools as a class are not as bright mentally as the children of the metropolis or of the northern parts of Victoria. They state that their opinions are supported by examination results. As other factors may play a part, it would be unwise to assume without reservations that the apparent mental lethargy is a direct result of the lack of thyreoid secretion. It is certainly a fact to be kept on record, but the experience of future generations will be necessary before an authoritative opinion could be given. At the Lucknow school one child was seen that exhibited evidence of hypothyreoidism and he approached the cretin type.

Inquiry amongst the practising doctors and a review of the admissions to the Sale and Bairnsdale Hospitals gave no evidence of goîtrous disabilities. The Bairnsdale records were personally inspected for a ten-year period and no case entry was found of any disturbance of thyreoid secretion whilst the Secretary of the Gippsland Hospital at Sale reports that during the last two years two patients only with exophthalmic goître were treated in hospital, one a girl aged seventeen and one a woman aged fifty-five years.

The conclusions are:

1. In Gippsland amongst school children of either sex endemic goître of a simple parenchymatous type is prevalent.

2. There is no associated undue prevalence of toxic goître.

3. There is some evidence that the thyreoid hyperplasia may be an effort to counteract a state of hypothyreoidism. Several generations must pass before definite evidence in this respect is forthcoming. Investigations are in progress as to the iodine content of water and food.

THE CLINICAL IMPORTANCE OF THE BASAL METABOLIC RATE IN GOITRE.

BY FRED. T. BOWERBANK, O.B.E., M.D., M.R.C.P. (Edinburgh),
Wellington.

ALTHOUGH the basal metabolic rate was described by von Noorden in 1907 in his "Metabolism and Practical Medicine" and by Magnus Levy about the same time, it is only within the last few years that its importance has been accepted by a majority of the medical profession in Britain and America. Delay in the recognition of this method has been partly due to exaggerated claims made by enthusiasts, but with increasing knowledge of the functions and correlation of the various endocrine glands it is now generally recognized that over or under activity of the thyreoid gland is the chief and most important factor responsible for changes in the metabolic rate. In hypopituitarism Beaumont and Dodds[1] state that there is only a slight decrease in the metabolic rate and that a differential diagnosis may be made by absence of signs of

myxœdema and by the injection of pituitrin. Also that hyperpituitarism, by enhancing the effect of the thyreoid gland, may slightly increase the metabolic rate.

Adrenalin[2] may increase the metabolic rate, but such effect is temporary and lasts only a few hours, whereas that of thyroxin lasts for days.

In my paper today, therefore, I shall deal only with the basal metabolic rate in its relation to disease of the thyreoid gland.

In America and more especially in the Mayo Clinic which I visited in 1925, and in certain of the British clinics, so much importance is attributed to this method that no diagnosis of hyperthyreoidism is accepted until it has been proved by the basal metabolic rate test and that subsequent medical and surgical treatment is controlled by this means. In Edinburgh Professor Murray Lyon, assisted by Dr. Whitridge Davies, has done much in improving the technique and it was while working in his biochemical laboratory in 1925 that I became convinced of the great value of this test, more especially in those cases, types of which I shall elaborate later in my paper. In New Zealand I regret to say very little has been done, although I understand that at the Dunedin Hospital it is being increasingly made use of as an aid in diagnosis *et cetera*. At the annual meeting of the New Zealand Branch of the British Medical Association held in Nelson in 1926 goître was one of the chief subjects for discussion and the basal metabolic test was not even mentioned, yet all forms of goître are extremely common in New Zealand and the mortality, surgical and medical, is not only unduly high, but there is little evidence that it is decreasing. From 1916 to 1925 there has been a steady increase in the mortality rates from diseases of the thyreoid gland. In 1916 the total number of deaths of males was five and of females thirty-five. In 1925 the total number of deaths of males was twelve and of females sixty-two. Taking the thyreoid diseases treated in public hospitals in New Zealand over the same period, in 1916 there were twenty-nine in males and one hundred and fifty-one in females, that is a total of one hundred and eighty, and in 1925 eighty-one in males and four hundred and twenty-two in females, a total of five hundred and three. These figures do not represent patients, but admissions and readmissions and yet the deaths in public hospitals in New Zealand from disease of the thyreoid gland in 1916 were three of males and ten of females, that is a total of thirteen, and in 1925 five of males and twenty-seven of females, that is a total of thirty-two.[3] For this reason alone any method whereby the death rate can be reduced, should not be neglected. It has been and still is with a few "die

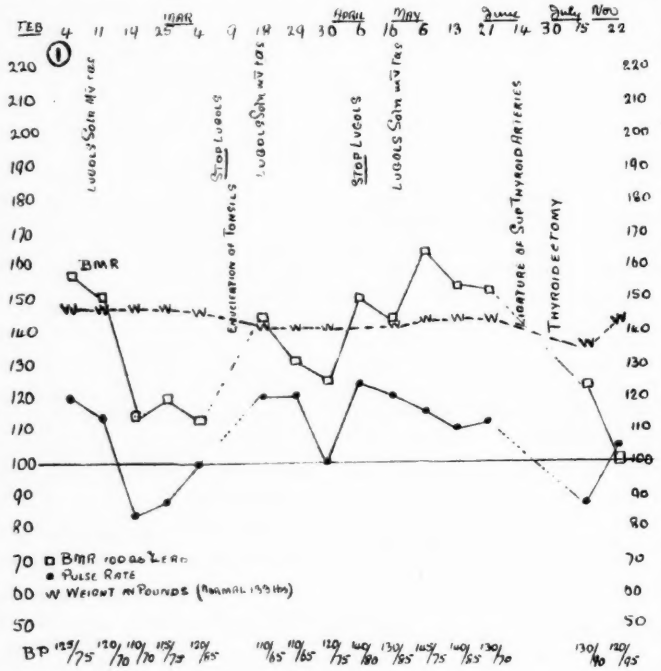

CHART I.

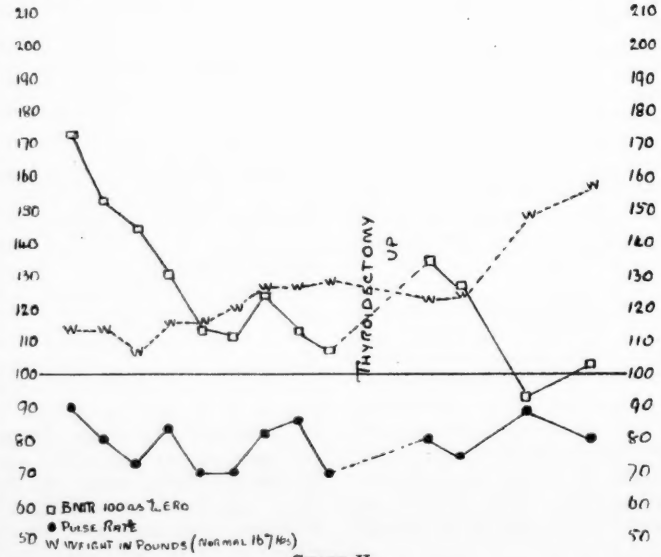

CHART II.

hards" a boast that diagnosis and treatment can be estimated by clinical observation alone. Would any conscientious surgeon operate on a patient for renal calculus or cholelithiasis or gastric ulcer before calling to his aid the various instrumental methods of diagnosis, such as X ray examination, test meals and so forth?

The importance of the basal metabolic rate from the clinical point of view is firstly in diagnosis, secondly to show the progress of the disease and the result of medical or surgical treatment and thirdly when it is advisable for the surgeon to operate and the extent of the operation.

Firstly the diagnosis of hyperthyreoidism may be extremely difficult. A few years ago there were certain so-called cardinal signs and symptoms, but now, with increasing knowledge, goître has been differentiated into several varieties and even the term exophthalmic goître is misleading, owing to the fact that exophthalmos is not invariably present. The size of the thyreoid gland is no indication of the severity or even activity of the gland. Some of the patients with the most severe forms of hyperthyreoidism have a thyreoid which is not visible. On the other hand a patient may have an enlarged thyreoid with such symptoms as nervousness, loss of weight, lassitude and so on and yet the condition is not due to the thyreoid at all. Neurasthenia, for instance, simulates hyperthyreoidism and *vice versa*. Both have common varietes and even symptoms and it may be impossible by ordinary clinical observation to differentiate. The treatment in these two conditions is totally different and much time may be wasted and much suffering may be borne by the patient before a correct diagnosis has been made.

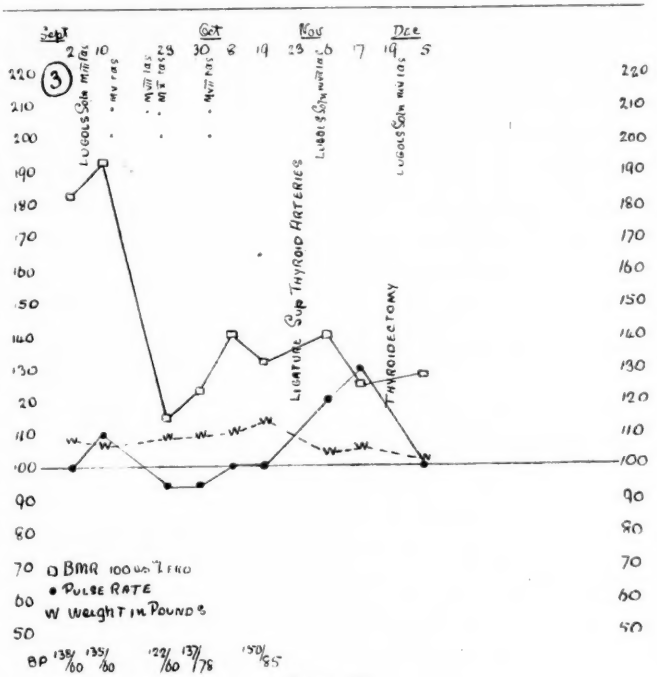

Chart III.

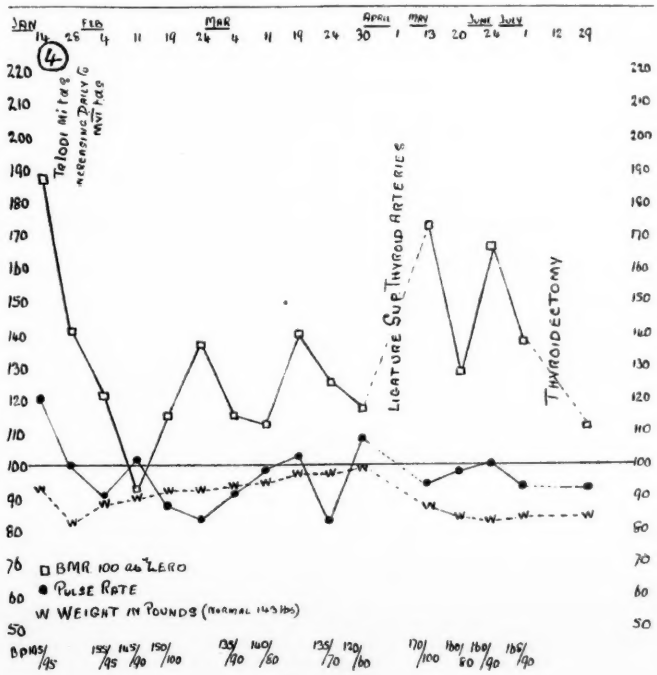

Chart IV.

Within the last few months I have seen several patients of this class. Two were returned soldiers who for years had been treated for neurasthenia and had even spent many months at Hanmer without benefit. The thyreoid gland in each was barely visible and even on palpation little change was detected and yet the basal metabolic rate was raised to 125 (100 as zero) in one and to 134 (100 as zero) in the other. I advised operation: both had a thyreoidectomy done. In both the basal metabolic rates fell to normal three weeks after the operation and the medical men who sent them to me, report complete recovery. In other cases the endocrine glands may be primarily at fault. One patient I tested some months ago had an enlarged right lobe. The medical man, a well known authority on goître in Wellington, was not certain whether the condition was a thyreotoxicosis or not and called in another medical man who diagnosed toxic adenoma and suggested operation. The first medical man thereupon asked me to estimate the basal metabolic rate and tests on two consecutive days revealed no increase in the metabolic rate. A

diagnosis of endocrine imbalance was made with adrenalin insufficiency as the probable exciting factor. Treatment was carried out on these lines and I understand that improvement has been uninterrupted. An operation in this case would have been extremely hazardous and under the circumstances useless.

The effort syndrome (or "D.A.H." as it was wrongly termed) is another condition which might be mistaken for hyperthyreoidism and undoubtedly many cases of hyperthyreoidism have been included under that diagnosis.

For instance, take the young woman working in a shop or factory all day, and going to the picture theatre or cabaret each night. She frequently consults a medical man, complaining of palpitation, loss of weight and so on. Then again there is the patient with the toxic type, with septic tonsils or teeth, complaining of lassitude, loss of weight, palpitation and a slightly enlarged thyreoid gland; in other words, a secondary hyperthyreoidism. Removal of the septic focus may or may not cure the condition, but treatment of the hyperthyreoidism alone certainly will not. On the lantern slides I shall show later two cases to illustrate this. In another case I was asked to see a probationer at the Wellington Hospital. She complained of palpitation, lassitude, loss of weight and her thyreoid gland was slightly enlarged. She gave a history of scarlet fever three months before. Repeated metabolic tests revealed a normal basal metabolic rate.

Glycosuria is not at all uncommon in hyperthyreoidism and in some cases may dominate the picture to such an extent that thyreotoxicosis as the primary factor may be overlooked. To illustrate this a man was admitted to the Wellington Hospital; his condition had been diagnosed as a case of *diabetes mellitus*. He had been treated for about two months for this condition. He was emaciated and gave the appearance generally of advanced diabetes or malignant disease. He had prominent eyes, but no definite evidence of the usual accompanying signs, such as von Graefe's or Stellwag's. On inquiry his wife stated that he had always prominent eyes. The thyreoid gland was not visible and on palpation did not appear to be enlarged. A glucose tolerance test was made and yielded a normal curve. A barium meal was given and X ray photographs showed nothing abnormal in the abdomen. The basal metabolic rate was then estimated and an increase to 173 (100 as zero) was ascertained. I shall later show a chart of this case showing the progress made under treatment for hyperthyreoidism.

In the second place it shows the progress of the disease and the result of medical and surgical treatment.

(To be Continued.)

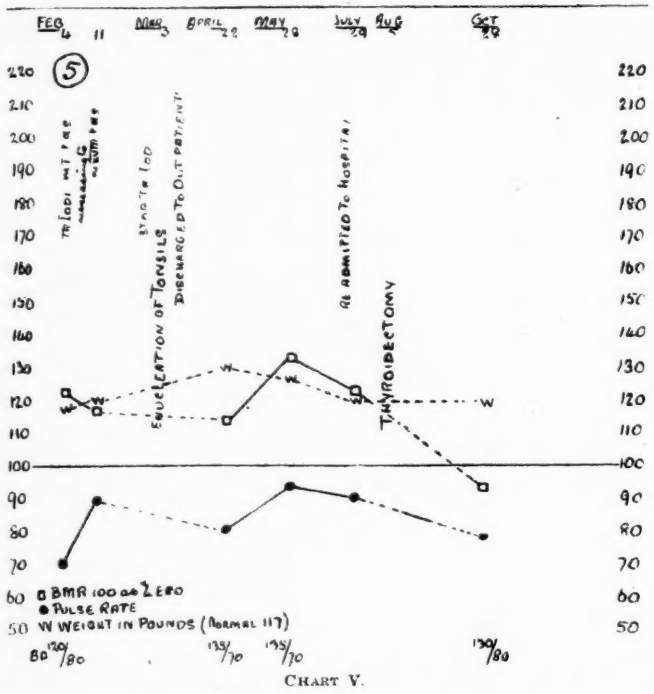

CHART V.

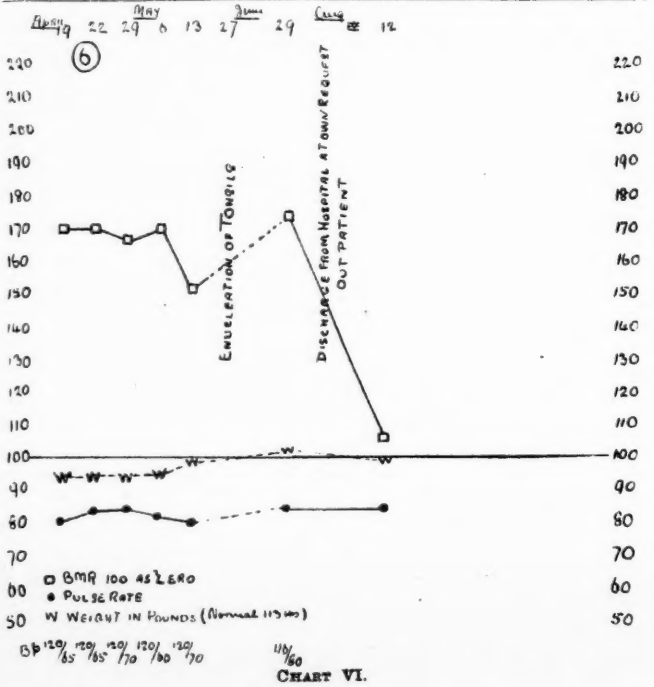

CHART VI.

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